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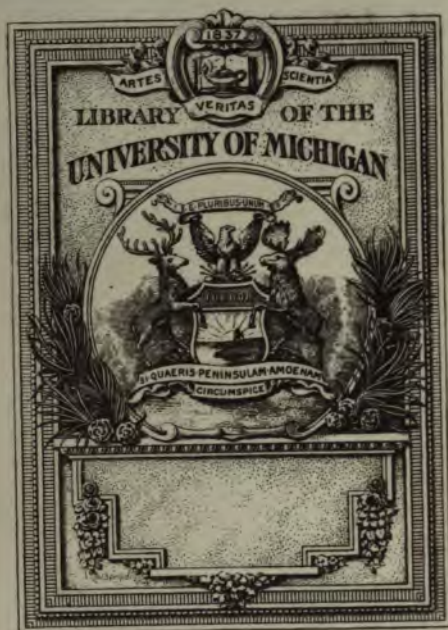
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# MEDICO-CHIRURGICAL TRANSACTIONS

PUBLISHED BY

THE ROYAL  
MEDICAL AND CHIRURGICAL SOCIETY  
OF  
LONDON

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VOLUME THE EIGHTY-FIRST

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*November, 1898.*

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ROYAL  
MEDICAL AND CHIRURGICAL SOCIETY  
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- 1805 WILLIAM SAUNDERS, M.D.
- 1808 MATTHEW BAILLIE, M.D.
- 1810 SIR HENRY HALFORD, BART., M.D., G.C.H.
- 1813 SIR GILBERT BLANE, BART., M.D.
- 1815 HENRY CLINE
- 1817 WILLIAM BABINGTON, M.D.
- 1819 SIR ASTLEY PASTON COOPER, BART., K.C.H.
- 1821 JOHN COOKE, M.D.
- 1823 JOHN ABERNETHY
- 1825 GEORGE BIRKBECK, M.D.
- 1827 BENJAMIN TRAVERS
- 1829 PETER MARK ROGET, M.D.
- 1831 SIR WILLIAM LAWRENCE, BART.
- 1833 JOHN ELLIOTSON, M.D. (First President of the Society after its Incorporation as the Royal Medical and Chirurgical Society of London in 1834).
- 1835 HENRY EARLE
- 1837 RICHARD BRIGHT, M.D.
- 1839 SIR BENJAMIN COLLINS BRODIE, BART.
- 1841 ROBERT WILLIAMS, M.D.
- 1843 EDWARD STANLEY
- 1845 WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
- 1847 JAMES MONCRIEFF ARNOTT
- 1849 THOMAS ADDISON, M.D.
- 1851 JOSEPH HODGSON
- 1853 JAMES COPLAND, M.D.
- 1855 CÆSAR HENRY HAWKINS
- 1857 SIR CHARLES LOCOCK, BART., M.D.
- 1859 FREDERIC CARPENTER SKEY
- 1861 BENJAMIN GUY BABINGTON, M.D.
- 1863 RICHARD PARTRIDGE
- 1865 SIR JAMES ALDERSON, M.D.
- 1867 SAMUEL SOLLY
- 1869 SIR GEORGE BURROWS, BART., M.D.
- 1871 THOMAS BLIZARD CURLING
- 1873 CHARLES JAMES BLASIUS WILLIAMS, M.D.
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- 1877 CHARLES WEST, M.D.
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- 1881 ANDREW WHYTE BARCLAY, M.D.
- 1882 JOHN MARSHALL
- 1884 SIR GEORGE JOHNSON, M.D.
- 1886 GEORGE DAVID POLLOCK
- 1888 SIR EDWARD HENRY SIEVEKING, M.D.
- 1890 TIMOTHY HOLMES
- 1892 SIR ANDREW CLARK, BART., M.D.  
(*Sir Andrew Clark died 6th November, 1893, and Dr. W. S. Church, Senior [Medical] Vice-President, officiated as President until the following 1st March, 1894.*)
- 1894 JONATHAN HUTCHINSON
- 1896 WILLIAM HOWSHIP DICKINSON, M.D.
- 1898 THOMAS BRYANT

## HONORARY FELLOWS

(Limited to Twelve.)

### *Elected*

- 1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S.,  
late Director of the Natural History Department,  
British Museum, Cromwell road.
- 1887 FOSTER, MICHAEL, M.D., LL.D., F.R.S., Professor of  
Physiology in the University of Cambridge.
- 1883 FRANKLAND, SIR EDWARD, K.C.B., M.D., D.C.L., Ph.D.,  
F.R.S., Associate Member of the Academy of Sciences  
of France; The Yews, Reigate Hill, Reigate.
- 1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., G.C.S.I., D.C.L.,  
LL.D., F.R.S., Corresponding Member of the Academy  
of Sciences of France; The Camp, Sunningdale.
- 1896 KELVIN, LORD, G.C.V.O., F.R.S., Pres.R.S.E., D.C.L.,  
LL.D., &c., Glasgow.
- 1878 LUBBOCK, The Right Hon. SIR JOHN, Bart., M.P., D.C.L.  
LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.
- 1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D.,  
Sc.D., F.R.S., Lucasian Professor of Mathematics in the  
University of Cambridge; Lensfield Cottage, Cam-  
bridge.
- 1887 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Pro-  
fessor of Anatomy in the University of Edinburgh;  
6, Eton Terrace, Edinburgh.

## FOREIGN HONORARY FELLOWS

(Limited to Twenty.)

### *Elected*

- 1878 BACCELLI, GUIDO, M.D., Rome.  
1896 VON BERGMANN, ERNST, Berlin.  
1887 BILLINGS, JOHN S., M.D., D.C.L. Oxon., New York.  
1896 CZEERNY, VINCENT, M.D., Heidelberg.  
1896 ERB, WILHELM, M.D., Professor of Clinical Medicine,  
Heidelberg.  
1887 VON ESMARCH, His Excellency FRIEDRICH, M.D., Kiel.  
1896 FOURNIER, ALFRED, M.D., Paris.  
1896 GERHARDT, CARL, M.D., Berlin.  
1896 KOCH, ROBERT, M.D., Berlin.  
1896 KOCHER, THEODORE, M.D., Berne.  
1868 KÖLLIKER, ALBERT, Würzburg.  
1896 LAVERAN, A., M.D., Paris.  
1896 MARIE, PIERRE, M.D., Paris.  
1896 MITCHELL, SAMUEL WEIR, M.D., Philadelphia.  
1896 MIRZA-ALI, M.D., Teheran.  
1856 VIRCHOW, RUDOLF, M.D., LL.D., Berlin.

# FELLOWS

OF THE

## ROYAL MEDICAL AND CHIRURGICAL SOCIETY

### OF LONDON

#### EXPLANATION OF THE ABBREVIATIONS

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V.P.—Vice-President.	<i>Sci. Com.</i> —Member of a Scientific Committee.
T.—Treasurer.	<i>Ho. Com.</i> —Member of House Committee.
L.—Hon. Librarian.	<i>Lib. Com.</i> —Member of Library Committee.
S.—Hon. Secretary.	<i>Bldg. Com.</i> —Member of Building Committee.
	<i>Dis. Com.</i> —Member of Discussions Committee.

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[N.B.—Fellows are reminded that they are, themselves, responsible for the correctness of the descriptions in the following lists, and it is particularly requested that any change of Title, Appointment, or Residence may be communicated to the Hon. Secretaries before the 1st of July in each year.]

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1898 AARONS, S. JERVOIS, M.D., 15, Devonshire place, Portland place.

1877 Abercrombie, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. C. 1896-8. *Referee*, 1898. *Trans.* 2.

*Elected*

- 1885 **ABRAHAM, PHINEAS S., M.A., M.D.**, Dermatologist to the West London Hospital, Assistant Surgeon to Hospital for Diseases of the Skin, Blackfriars; 2, Henrietta street, Cavendish square.
- 1885 **ACLAND, THEODORE DYKE, M.D.**, Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square.
- 1852 **Adams, WILLIAM**, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopaedic Hospital; 7, Loudoun road, St. John's Wood. C. 1873-4. *Trans.* 3.
- 1879 **ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed.**, Senior Physician to the Westminster Hospital; 5, Chandos street, Cavendish square. C. 1898—. *Referee*, 1897.
- 1890 **ALLINGHAM, HERBERT WILLIAM**, Surgeon to the Great Northern Hospital; Assistant Surgeon to St. George's Hospital; 25, Grosvenor street, Grosvenor square.
- 1863 **Althaus, JULIUS, M.D.**, Consulting Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 26, Queen Anne street, Cavendish square. *Trans.* 2.
- 1888 **ANDERSON, JOHN, M.D., C.I.E.**, Physician to the Seamen's Hospital, Greenwich; Lecturer on Tropical Medicine at St. Mary's Hospital Medical School; 9, Harley street, Cavendish square.
- 1890 **ANDERSON, WILLIAM**, Surgeon to St. Thomas's Hospital; Professor of Anatomy to the Royal Academy of Arts; 2, Harley street, Cavendish square. *Lib. Com.* 1896-8.
- 1891 **ANDREWES, FREDERICK WILLIAM, M.D.**, Highwood, Hampstead Lane, Highgate.
- 1888 **ARKLE, CHARLES, M.D.**, Assistant Physician to Charing Cross Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton; 66, Wimpole street, Cavendish square.

*Elected*

- 1893 BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.
- 1891 BAKER, CHARLES ERNEST, M.B., 5, Gledhow gardens, South Kensington.
- 1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital; 12, Upper Wimpole street, Cavendish square.
- 1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 3.
- 1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895-7. *Referee*, 1897—. *Trans.* 7.
- 1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*; Physician-in-Ordinary to H. M.'s Household; Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Referee*, 1896—. *Trans.* 2.
- 1893 BARRETT, HOWARD, 49, Gordon square.
- 1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.
- 1896 BARTON, JAMES KINGSTON, 2, Courtfield road, Gloucester road, South Kensington.
- 1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12. *Pro.* 1.

*Elected*

- 1868 **Bastian**, HENRY CHARLTON, M.A., M.D., F.R.S., Emeritus Professor of the Principles and Practice of Medicine and of Clinical Medicine in University College, London; Consulting Physician to University College Hospital and Physician to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886-96. *Trans.* 3.
- 1890 **BATEMAN**, WILLIAM A. F., Bridge House, Richmond, Surrey.
- 1891 **BATTEN**, FREDERICK E., M.D., B.C., 124, Harley street.
- 1875 **BEACH**, FLETCHER, M.B., Winchester House, Kingston Hill [64, Welbeck street].
- 1883 **BEALE**, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.
- 1862 **BEALE**, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.
- 1897 **BEDDARD**, A. P., M.B., 44, Seymour street.
- 1880 **BEEVOR**, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 33, Harley street, Cavendish square. *Referee*, 1896—. *Trans.* 1
- 1883 **BENNETT**, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at the Middlesex Hospital; Lecturer on Dental Surgery and Pathology at the Dental Hospital of London; 17, George street, Hanover square.
- 1877 **BENNETT**, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93. *Trans.* 4.

*Elected*

- 1897 **BERKELEY, COMYNS, M.B., B.C.**, Physician to Out-Patients, Chelsea Hospital for Women; 53, Wimpole street.
- 1845 **Berry, EDWARD UNWIN**, 17, Sherriff road, West Hampstead.
- 1885 **BERRY, JAMES, B.S.**, Surgeon to, and Lecturer on Surgery at, the Royal Free Hospital; Demonstrator of Practical Surgery, St. Bartholomew's Hospital; 60, Welbeck street, Cavendish square.
- 1893 **BIDWELL, LEONARD A.**, Senior Assistant Surgeon to the West London Hospital; 59, Wimpole street.
- 1856 **Bird, WILLIAM**, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- 1851 **Birkett, JOHN, F.L.S.**, Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; 1, Sussex gardens. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.
- 1881 **BISS, CECIL YATES, M.D.**, Physician to Out-Patients and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.
- 1897 **BLACKER, G. F., M.D.**, 20, Weymouth street.
- 1865 **Blandford, GEORGE FIELDING, M.D.**, Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4. V.P. 1898—.
- 1891 **BOKENHAM, THOMAS JESSOPP**, 10, Devonshire street, Portland place.
- 1882 **BOWLBY, ANTHONY ALFRED**, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 8.
- 1870 **Bowles, ROBERT LEAMON, M.D.**, 16, Upper Brook street, Grosvenor square. C. 1897—. *Sci. Com.* 1896—. *Trans.* 2.



*Elected*

- 1886 **BOXALL, ROBERT, M.D.**, Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 40, Portland place.
- 1884 **BOYD, STANLEY, M.B.**, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; Consulting Surgeon to the New Hospital for Women; 134, Harley street, Cavendish square. *Referee*, 1895—. *Trans.* 1.
- 1890 **BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S.**, Physician to University College Hospital; 60, Wimpole street. *Trans.* 1.
- 1883 **BRADSHAW, JAMES DIXON, M.B.**, 36, Avenue road, Regent's park.
- 1897 **BRAILEY, WILLIAM ARTHUR, M.D.**, 11, Old Burlington street.
- 1890 **BRINTON, ROLAND DANVERS, M.D.**, 8, Queen's Gate terrace.
- 1898 **Broadbent, J. F. H., M.D.**, 35, Seymour street.
- 1868 **Broadbent, SIR WILLIAM HENRY, Bart., M.D., F.R.S., LL.D.**, Physician in Ordinary to H.R.H. the Prince of Wales; Physician-Extraordinary to Her Majesty the Queen; Consulting Physician to St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891-7. *Trans.* 5.
- 1851 **Brodhurst, BERNARD EDWARD**, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member, Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 21, Portland place. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.
- 1872 **BRODIE, GEORGE BERNARD, M.D.**, Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

*Elected*

- 1880 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park avenue.
- 1881 BROWNE, OSWALD AUCHINLECK, M.A., M.D., Physician to the Royal Hospital for Diseases of the Chest and to the Metropolitan Hospital; 7, Upper Wimpole street.
- 1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Consulting Physician to the Hospital for Consumption Brompton; 23, Harley street. C. 1892, 1897—. S. 1893-5. *Sci. Com.* 1889—. *Ho. Com.* 1898—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.
- 1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Physician to, and Lecturer on Pharmacology and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 2.
- 1898 BRYANT, J. H., M.D., Assistant Physician to Guy's Hospital; 8, St. Thomas's street, London bridge.
- 1860 BRYANT, THOMAS, M.Ch., *President*, Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V.P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.
- 1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 5, Clarges street, Piccadilly.
- 1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Surgeon to King's College Hospital and Paddington Green Children's Hospital; 86, Harley street, Cavendish square.
- 1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Out-Patient Surgeon, Samaritan Free Hospital for Women and Children, Soho; Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

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- 1873 BUTLIN, HENRY TRENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4. *Pro.* 1.
- 1896 BUTTAR, CHARLES, M.D., 2, Princes square, Bayswater. *Pro.* 1.
- 1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Consulting Anæsthetist to the National Hospital for the Paralysed and Epileptic, Queen square, and Anæsthetist to the London Dental Hospital; 82, Mortimer street, Cavendish square.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.
- 1885 CAHILL, JOHN, M.D., Surgeon to the Hospital of St. John and St. Elizabeth; 12, Seville street, Lowndes square.
- 1893 CALEY, HENRY ALBERT, M.D., Physician in charge of Out-patients, Lecturer in Materia Medica and Therapeutics, and Senior Medical Tutor, St. Mary's Hospital; 24, Upper Berkeley street, Portman square.
- 1887 CALVERT, JAMES, M.D., The Warden's House, St. Bartholomew's Hospital.
- 1897 CANTLIE, JAMES, M.B., 46, Devonshire street.
- 1888 CARLESS, ALBERT, M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.
- 1896 CARR, J. WALTER, M.D., Assistant Physician to the Royal Free Hospital; 19, Cavendish place. *Trans.* 1.
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.
- 1898 CARTER, H. RONALD, 11, Leonard place, Kensington.
- 1853 CARTER, ROBERT BRUDENELL, Consulting Ophthalmic Surgeon to St. George's Hospital; 31, Harley street, Cavendish square, and Kenilworth, Clapham common. *Trans.* 1.

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- 1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street.
- 1871 CAYLEY, WILLIAM, M.D., Physician to the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.
- 1879 CHAMPNEYS, FRANCIS HENRY, M.D., Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. C. 1898—. *Referee*, 1891-8. *Lib. Com.* 1885-8. *Trans.* 8.
- 1868 Cheadle, WALTER BUTLER, M.D., *Trustee*; Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.
- 1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. C. 1897—. *Referee*, 1894-7. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.
- 1890 CHILDS, CHRISTOPHER, M.D., 10, Manchester square.
- 1896 CHRISTOPHERSON, JOHN BRIAN, M.B., B.C., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; St. Bartholomew's Hospital.
- 1866 Church, WILLIAM SELBY, M.D., *Hon. Treasurer*, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81. *Ho. Com.* 1898—.
- 1879 CLARK, ANDREW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

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- 1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 3, Chandos street, Cavendish square.
- 1890 CLARKE, JAMES JACKSON, M.B., Assistant Surgeon to the North-West London and City Orthopædic Hospitals; 9, Old Cavendish street, Cavendish square.
- 1848 CLARKE, JOHN, M.D., 42, Hertford street, Mayfair. C. 1866.
- 1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital, 51, Harley street, Cavendish square. *Trans.* 1.
- 1879 CLUTTON, HENRY HUGH, M.B., M.C., Senior Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 2, Portland place. C. 1897—. *Dis. Com.* 1897-8. *Referee*, 1896-7. *Trans.* 3.
- 1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.
- 1897 COLMAN, W. S., M.D., 22, Wimpole street.
- 1865 COOPER, ALFRED, Surgeon in Ordinary to H.R.H. the Duke of Saxe Coburg-Gotha; Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital; 9, Henrietta street, Cavendish square.
- 1898 CORFIELD, W. H., M.D., Professor of Hygiene and Public Health at University College, London; Honorary Sanitary Adviser to University College Hospital; Medical Officer of Health for St. George's, Hanover square; 19, Savile row, and Whindown, Bexhill, Sussex.
- 1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.
- 1860 COUPER, JOHN, Consulting Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

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- 1877 COUPLAND, SIDNEY, M.D., Commissioner in Lunacy ; late Physician to, and Lecturer on Medicine at, the Middlesex Hospital ; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee*, 1892-3. *Ho. Com.* 1895-8.
- 1862 COWELL, GEORGE, Consulting Surgeon to, and Vice-President of, the Westminster Hospital ; Consulting Surgeon to, and Treasurer of, the Royal Westminster Ophthalmic Hospital ; 19, Harley street, Cavendish square. C. 1882-3.
- 1897 CRAWFURD, RAYMOND H. PAYNE, M.D., 71, Harley street.
- 1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy ; 61, Carlisle place Mansions, Victoria street.
- 1874 CRIPPS, WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital ; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.
- 1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital ; late Physician to the East London Hospital for Children ; 121, Harley street, Cavendish square. *Trans.* 3.
- 1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital ; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.
- 1890 CROWLE, THOMAS HENRY RICKARD, 56, Harley street, Cavendish square.
- 1888 CULLINGWORTH, CHARLES JAMES, M.D., D.C.L., Obstetric Physician to St. Thomas's Hospital ; 14, Manchester square. *Referee*, 1896—.
- 1879 CUMBERBATCH, A. ELKIN, M.B., Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic ; 80, Portland place.

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- 1873 CURNOW, JOHN, M.D., Professor of Clinical Medicine in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 9, Wimpole street, Cavendish square. *Referee*, 1884-97.
- 1898 CURRIE, A. STARK, M.D., 81, Queen's road, Finsbury park.
- 1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to, and Lecturer in Midwifery at, St. George's Hospital, and Physician to the General Lying-in Hospital; 18, Grosvenor street, Grosvenor square.
- 1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. C. 1896-7. *Trans.* 3.
- 1891 DALTON, NORMAN, M.D., Physician to King's College Hospital; Professor of Pathological Anatomy in King's College, London; 4, Mansfield street, Cavendish square.
- 1896 DAUBER, JOHN HENRY, M.B., B.Ch., Assistant Physician to the Hospital for Women, Soho square; 29, Charles street, Berkeley square.
- 1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.
- 1889 DEAN, HENRY PERCY, M.S., Assistant Surgeon to the London Hospital; 69, Harley street, Cavendish square.
- 1878 DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 5.
- 1891 DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital; 91, Harley street.
- 1894 DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street.

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- 1859 **Dickinson, WILLIAM HOWSHIP, M.D.**, Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. P. 1896-8. C. 1874-5. V. P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 16.
- 1891 **Dickinson, WILLIAM LEE, M.D.**, Assistant Physician to St. George's Hospital and to the Hospital for Sick Children; 9, Chesterfield street, Mayfair.
- 1889 **DODD, HENRY WORK**, Surgeon to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the Royal Free Hospital and to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.
- 1888 **DONELAN, JAMES, M.B., M.C.**, Physician to the Italian Hospital, Queen square; 2, Upper Wimpole street, Cavendish square.
- 1877 **DORAN, ALBAN HENRY GRIFFITHS**, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-4. *Lib. Com.* 1891-3. *Referee*, 1898—. *Trans.* 3.
- 1891 **DOVE, PERCY W., M.B.**, 80, Crouch hill.
- 1896 **DOWNES, JOSEPH LOCKHART, M.B., C.M.**, 271, Romford road.
- 1879 **DREWITT, F. G. DAWTREY, M.D.**, Physician to the West London Hospital; 2, Manchester square.
- 1893 **DRYSDALE, JOHN H., M.B.**, 25, Welbeck street, Cavendish square.
- 1865 **Duckworth, SIR DYCE, M.D., LL.D.**, Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-97. *Trans.* 2.



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- 1876 DUDLEY, WILLIAM LEWIS, M.D., Senior Physician to the City Dispensary; 149, Cromwell road, South Kensington.
- 1871 DUKE, BENJAMIN, M.D., Windmill House, Clapham Common.
- 1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.
- 1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.
- 1887 DUNN, HUGH PERCY, Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.
- 1898 DUNN, L. A., M.S., The College, Guy's Hospital.
- 1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.
- 1894 DURHAM, HERBERT EDWARD, M.B., 82, Brook street, Grosvenor Square. *Trans.* 2.
- 1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester terrace, Hyde Park. C. 1892-3.
- 1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.
- 1893 ECCLES, WILLIAM McADAM, M.S., 124, Harley street.
- 1891 EDDOWES, ALFRED, M.D., 28, Wimpole street.
- 1898 EDKINS, J. S., 4, Park hill road, Hampstead.
- 1898 EDMUNDS, P. J., 5, Great Marlborough street, Regent street.
- 1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 3.

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- 1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; Senior Assistant Surgeon to St. Mark's Hospital for Diseases of the Rectum; 55, Harley street, Cavendish square.
- 1898 EVANS, WILLMOTT, 13, Taviton street, Gordon square.
- 1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. C. 1897—. *Trans.* 4.
- 1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital and to the Belgrave Hospital for Children; 33, Curzon street, Mayfair. C. 1895-7. *Lib. Com.* 1897—. *Sci. Com.* 1889—. *Trans.* 1. *Pro.* 1.
- 1872 FAYRE, SIR JOSEPH, Bart., K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; late Physician to the Secretary of State for India in Council, and President of the Medical Board at the India Office; 16, Devonshire street, Portland place. C. 1888. *Referee*, 1881-7.
- 1898 FENWICK, E. HURRY, 14, Savile row.
- 1863 FENWICK, SAMUEL, M.D., Consulting Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-96. *Trans.* 4.
- 1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuro-pathology in King's College, London, and Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891-6. C. 1896-8. *Dis. Com.* 1896—. *Trans.* 2.
- 1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.

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- 1891 FLETCHER, HERBERT MORLEY, M.D., 98, Harley street, Cavendish square.
- 1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.
- 1883 FOWLER, JAMES KINGSTON, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.
- 1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Diseases of the Skin to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* i.
- 1871 FRANK, PHILIP, M.D., 3, Elvaston place, South Kensington.
- 1896 FREYER, P. J., M.D., M.Ch., Surgeon to St. Peter's Hospital for Stone and Urinary Diseases; 46, Harley street, Cavendish square. *Trans.* 1.
- 1898 FRIPP, A. DOWNING, M.S., Surgeon in Ordinary to H.R.H. the Prince of Wales; Assistant Surgeon to Guy's Hospital; 19, Portland place.
- 1898 FROST, W. ADAMS, 30, Cavendish square.
- 1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.
- 1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.
- 1894 FURNIVALL, PERCY, 39, Welbeck street, Cavendish square.
- 1874 GALABIN, ALFRED LEWIS, M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole-st., Cavendish square. C. 1892. *Referee*, 1882-91, 1896—. *Lib. Com.* 1883-4. *Trans.* 2.
- 1895 GALLOWAY, JAMES, M.D., 54, Harley street, Cavendish square.
- 1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

*Elected*

- 1865 **Gant, FREDERICK JAMES**, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park C. 1880-81. V.P. 1897—. *Referee*, 1886-97. *Lib. Com.* 1882-5. *Trans.* 3.
- 1854 **Garrod, SIR ALFRED BARING, M.D., F.R.S.**, Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 9.
- 1886 **GARROD, ARCHIBALD EDWARD, M.D.**, Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children, Great Ormond street; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Lib. Com.* 1896—. *Trans.* 5.
- 1887 **GAY, JOHN**, 119, Upper Richmond road, Putney.
- 1866 **Gee, SAMUEL JONES, M.D., Hon. Librarian; Chairman of Trustees for Debenture-holders**; Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Ho. Com.* 1898—. *Trans.* 1.
- 1885 **GELL, HENRY WILLINGHAM, M.B.**, 36, Hyde Park square.
- 1880 **GIBBONS, ROBERT ALEXANDER, M.D.**, Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place. C. 1896-7. *Trans.* 1.
- 1872 **GILBART-SMITH, THOMAS, M.D.**, Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.
- 1893 **GILES, ARTHUR EDWARD, M.D., B.Sc.**, Assistant Surgeon, Chelsea Hospital for Women; 37, Queen Anne street.
- 1894 **GILL, RICHARD**, 17, Wigmore street.

*Elected*

- 1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Librarian* ; Surgeon in Ordinary to Her Majesty's Household ; Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London ; Surgeon to the Hospital for Consumption, Brompton ; Consulting Surgeon to the North-Eastern Hospital for Children ; 19, Wimpole street, Cavendish square. S. 1892-4. L. 1895—. *Referee*, 1886-91. *Ho. Com.* 1898—. *Trans.* 9.
- 1870 Godson, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital ; 9, Grosvenor street, Grosvenor square.
- 1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital ; 12, Queen Anne street, Cavendish square. *Trans.* 1.
- 1897 GOODBODY, F. W., M.D., 35, Bedford Court Mansions, Bloomsbury.
- 1896 GOODALL, EDWARD WILBERFORCE, M.D., B.S., Eastern Hospital, Homerton.
- 1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital ; Consulting Physician to the Evelina Hospital for Sick Children ; 25, Portland place. *Lib. Com.* 1893-6.
- 1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital ; Surgeon to St. Mark's Hospital ; 17, Devonshire place, Upper Wimpole street.
- 1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.
- 1877 GOULD, ALFRED PEARCE, M.S., *Hon. Secretary*, Surgeon to the Middlesex Hospital ; 10, Queen Anne street, Cavendish square. C. 1892-3. S. 1898—. *Referee*, 1895-8. *Ho. Com.* 1892-8. *Lib. Com.* 1891. *Trans.* 2.
- 1891 Gow, WILLIAM J., M.D., Assistant Obstetric Physician to St. Mary's Hospital ; Obstetric Physician to the Royal Hospital for Women and Children ; Physician to Out-Patients, Queen Charlotte's Lying-in Hospital ; 27, Weymouth street, Portland place.

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- 1873 **Gowers, Sir William Richard, M.D., F.R.S.**, Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.
- 1892 **Grant, J. Dundas, M.A., M.D.**, 8, Upper Wimpole street, Cavendish square.
- 1898 **Granville, Alexander**, St. Bartholomew's Hospital.
- 1868 **Green, T. Henry, M.D.**, Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.
- 1885 **Griffith, Walter Spencer Anderson, M.D.**, Assistant Physician-Accoucheur, St. Bartholomew's Hospital; Physician to Queen Charlotte's Lying-in Hospital; 96, Harley street, Cavendish square.
- 1868 **Grigg, William Chapman, M.D.**, Senior Physician to Queen Charlotte's Lying-in Hospital; late Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.
- 1889 **Gubb, Alfred S., M.D.**, 29, Gower street.
- 1883 **Gunn, Robert Marcus, M.B.**, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the National Hospital for the Paralysed and Epileptic; 54, Queen Anne street, Cavendish square.
- 1890 **Guthrie, Leonard George, M.D., B.Ch.**, Physician to the Regent's Park Hospital for Epilepsy and Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.
- 1886 **Habershon, Samuel Herbert, M.D.**, Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.

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- 1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.
- 1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.
- 1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893-7.
- 1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.
- 1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.
- 1856 HARE, CHARLES JOHN, M.D., Consulting Physician to University College Hospital; late Professor of Clinical Medicine, University College; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-94. V.P. 1894-5. *Bldg. Com.* 1889-92.
- 1864 HARLEY, JOHN, M.D., F.L.S., Hon. Physician to St. Thomas's Hospital; Consulting Physician to the London Fever Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. V.P. 1895-7. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.
- 1893 HARLEY, VAUGHAN, M.D., 25, Harley street, Cavendish square.
- 1892 HAROLD, JOHN, 91, Harley street, Cavendish square.
- 1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 22, Queen Anne street, Cavendish square.
- 1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. C. 1894-5. V.-P. 1898—. *Trans.* 2.

*Elected*

- 1870 **HAWARD, J. WARRINGTON**, *Hon. Treasurer*; Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. V.P. 1894-5. T. (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (Sec.) 1889-92. *Ho. Com.* 1892—. *Trans.* 3.
- 1891 **HAWKINS, HERBERT PENNELL**, M.D., B.Ch., Physician to St. Thomas's Hospital; 56, Portland place.
- 1875 **HAYES, THOMAS CRAWFORD**, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Professor of Midwifery in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.
- 1860 **Hayward, HENRY HOWARD**, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.
- 1891 **HAYWARD, JOHN ARTHUR**, M.D., 17, Lingfield road, Wimbledon. *Pro.* 1.
- 1865 **Heath, CHRISTOPHER**, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.
- 1882 **HENSLEY, PHILIP JOHN**, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square. *Referee*, 1897—.
- 1877 **HERMAN, GEORGE ERNEST**, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Lib. Com.* 1898—. *Trans.* 1.
- 1877 **HERON, GEORGE ALLAN**, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.
- 1891 **HERRING, HERBERT T.**, M.B., B.S., 50, Harley street, Cavendish square.



*Elected*

- 1883 HERRINGHAM, WILMOT PARKER, M.D., Assistant Physician, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 2.
- 1893 HERSCHELL, GEORGE, M.D., 27, Queen Anne street, Cavendish square.
- 1887 HEWITT, FREDERIC WILLIAM, M.D., Anæsthetist to, and Instructor in Anæsthetics at, the London Hospital; Anæsthetist at the Dental Hospital of London; 14, Queen Anne street, Cavendish square. *Trans.* 2.
- 1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 52, Brook street, Grosvenor square. C: 1894-5. *Trans.* 2.
- 1890 HILL, G. WILLIAM, M.D., B.Sc., 28, Weymouth street.
- 1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 6, Sussex place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.* 1892-8. *Trans.* 8.
- 1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the West London Hospital; Examining Physician for Queen's Messengers, Foreign Office; 43, Green street, Park lane.
- 1898 HORDER, T. JEEVES, 29, Constantine road, Hampstead.
- 1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. *Referee*, 1897—. *Trans.* 1.
- 1896 HORTON-SMITH, PERCIVAL, M.D., 15, Upper Brook street. *Sci. Com.* 1897—. *Trans.* 1.
- 1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

*Elected*

- 1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.
- 1828 HULKE, S. BACKHOUSE, 162, Holland road, Kensington.
- 1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; Curator and Pathologist, Charing Cross Hospital; 103, Harley street.
- 1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital.
- 1897 HUTCHISON, R., M.D., Toynbee Hall, Whitechapel.
- 1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.
- 1888 HUTCHINSON, JONATHAN, Jun., Surgeon to the London Hospital; 1, Park crescent. *Trans.* 1.
- 1871 JACKSON, J. HUGHLINGS, M.D., LL.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.
- 1883 JACOBSON, WALTER HAMILTON ACLAND, M.Ch.Oxon., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Referee*, 1895—. *Lib. Com.* 1896—. *Trans.* 2.

*Elected*

- 1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens, and Pavilion, Melrose, N.B.
- 1897 JAMISON, ARTHUR ANDREW, M.D., 18, Lowndes street, Belgrave square.
- 1897 JENNER, LOUIS, M.B., 4A, Bloomsbury square.
- 1883 JESSOP, WALTER H. II., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.
- 1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, and 36, Finsbury pavement.
- 1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.
- 1884 JOHNSTON, JAMES, M.D., 53, Prince's square, Bayswater.
- 1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 61, Wimpole street, Cavendish square.
- 1896 JONES, L. VERNON, B.A., M.D., B.Ch., 7, Arlington street, St. James's.
- 1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.
- 1898 KEEP, A. CORRIE, M.D., C.M., 14, Gloucester place, Portman square.
- 1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.
- 1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital; 11, Harley street, Cavendish square.
- 1857 Kiallmark, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

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- 1881 KIDD, PERCY, M.D., Physician to the Hospital for Consumption, Brompton; Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.
- 1851 Kingdon, JOHN ABERNETHY, Consulting Surgeon to the Bank of England, Threadneedle street. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.
- 1896 LANE, JAMES ERNEST, 46, Queen Anne street, Cavendish square.
- 1884 LANE, WILLIAM ARBUTHNOT, M.S., Surgeon to Guy's Hospital and to the Hospital for Sick Children; 21, Cavendish square. *Trans.* 4.
- 1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.
- 1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 81, Harley street.
- 1865 Langton, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. V.P. 1895-7. *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95. *Trans.* 2.
- 1898 LATHAM, A. C., M.D., 13, Bruton street, Berkeley square.
- 1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.
- 1898 LAWFORD, J. B., 99, Harley street.
- 1888 LAWRENCE, LAURIE ASHER, 4, Queen Anne street.
- 1893 LAWSON, ARNOLD, Ophthalmic Surgeon to the Children's Hospital, Paddington Green; 12, Harley street, Cavendish square.

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- 1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.
- 1892 LAZARUS-BARLOW, WALTER SYDNEY, M.B., 55, Penn road villas, West Holloway. *Sci. Com.* 1892—. (Traveling.)
- 1892 LEADAM, WILLIAM WARD, M.D., 167, Gloucester terrace, Hyde Park.
- 1896 LEE, WILLIAM EDWARD, M.B., Santhapuram, Muswell hill road, Highgate.
- 1895 LEES, DAVID BRIDGE, M.D., 22, Weymouth street, Portland place. *Trans.* 2.
- 1895 LESLIE, ROBERT MURRAY, M.B., 53, Queen Anne street.
- 1897 LEVY, ALFRED G., M.D., 124, Barons Court road, West Kensington.
- 1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 72, Harley street, Cavendish square. *Trans.* 1.
- 1896 LEWIS, FREDERICK HENRY, M.B., St. Bartholomew's Hospital, and 71, The Drive, West Brighton.
- 1878 LISTER, LORD, D.C.L., LL.D., P.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.
- 1891 LITTLE, ERNEST MUIRHEAD, Surgeon to the National Orthopædic Hospital; 40, Seymour street, Portman square.
- 1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.
- 1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Lecturer on Surgical and Descriptive Anatomy at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 4.

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- 1897 LOW, HAROLD, 10, Evelyn gardens.
- 1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the Société de Chirurgie of Paris; 50, Wimpole street, Cavendish square. *Trans.* 2.
- 1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; 31, Weymouth street, Portland place. *Trans.* 1.
- 1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.
- 1898 LYSTER, C. R. C., Bolingbroke Hospital, Wandsworth common.
- 1873 MacCarthy, JEREMIAH, M.A., Surgeon to the London Hospital, late Lecturer on Surgery at the London Hospital Medical College; 1, Cambridge place, Victoria road, Kensington. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.
- 1867 MAC CORMAC, SIR WILLIAM, Bart., M.Ch., D.Sc., Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon and Emeritus Lecturer on Clinical Surgery to St. Thomas's Hospital; 13, Harley street. C. 1884-5. V.P. 1896-7. *Referee*, 1889-96. *Lib. Com.* 1895. *Trans.* 1.
- 1894 MACFADYEN, ALLAN, M.D., B.S., British Institute of Preventive Medicine, Chelsea bridge.
- 1898 McFADYEAN, JOHN, The Royal Veterinary College, Camden town.
- 1896 MACGREGOR, ALEXANDER, M.D., 7, Harley street.
- 1880 McHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Senior Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

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- 1873 **MACKELLAR, ALEXANDER OBERLIN, M.Ch.**, Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.
- 1881 **MACKENZIE, STEPHEN, M.D.**, Physician to the London Hospital; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.
- 1879 **MACLAGAN, THOMAS JOHN, M.D.**, Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.
- 1889 **MACLEHOSE, NORMAN MACMILLAN, M.B., C.M.**, 13, Queen Anne street, Cavendish square.
- 1876 **MACNAMARA, N. CHARLES**, Consulting Surgeon to the Westminster Hospital, and to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor street. *C.* 1891-2. *Referee*, 1884-90, 1895-7. *Lib. Com.* 1886-90.
- 1881 **MACREADY, JONATHAN FORSTER CHRISTIAN HORACE**, Surgeon to the Great Northern Hospital; 42, Devonshire street.
- 1880 **MADDICK, EDMUND DISTIN**, 2, Chandos street, Cavendish square.
- 1886 **MAGUIRE, ROBERT, M.D.**, Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.
- 1880 **MAKINS, GEORGE HENRY**, Assistant Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Referee*, 1898—. *Trans.* 1.
- 1885 **MALCOLM, JOHN DAVID, M.B., C.M.**, Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.

*Elected*

- 1890 **MANSON, PATRICK, M.D., C.M., LL.D.**, Physician to the Seamen's Hospital, Albert Docks ; 21, Queen Anne street, Cavendish square.
- 1855 **MARCEY, WILLIAM, M.D., F.R.S.**, Flowermead, Wimbledon Park. C. 1871. V.P. 1897—. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.
- 1867 **MARSH, F. HOWARD**, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital ; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.
- 1891 **MARTIN, HENRY CHARRINGTON, M.D.**, 27, Oxford square.
- 1884 **MARTIN, SIDNEY HARRIS COX, M.D., F.R.S.**, Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton ; Professor of Pathology, University College, London ; 10, Mansfield street, Portland place.
- 1892 **MASTERS, JOHN ALFRED, M.D.**, 57, Lexham gardens, Kensington.
- 1891 **MAY, WILLIAM PAGE, M.D., B.Sc.**, 49, Welbeck street.
- 1891 **MERCIER, CHARLES ARTHUR, M.B.**, Lecturer on Neurology and Insanity to Westminster Hospital ; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.
- 1880 **MEREDITH, WILLIAM APPLETON, M.B., C.M.**, Surgeon to the Samaritan Free Hospital for Women and Children ; 21, Manchester square. C. 1897-8. *Ho. Com.* 1898—. *Trans.* 1.
- 1897 **MERRY, WILLIAM JOSEPH COLLINGS, M.D., B.Ch.**, 1, Cleveland square, Hyde park.
- 1894 **MICHEL, ERNST, M.D.**, Surgeon to the German Hospital ; 6, West street, Finsbury circus. *Trans.* 2.
- 1893 **MILEY, MILES, M.B.**, 21, Belsize avenue, Hampstead.
- 1891 **MOLINE, PAUL, M.B.**, 42, Walton street, Chelsea.



*Elected*

- 1873 MOORE, NORMAN, M.D., *Hon. Secretary*, Assistant Physician and Lecturer on Medicine to St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. S. 1896—. *Referee*, 1886-90. *Sci. Com.* 1889—.
- 1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. C. 1895-7. *Dis. Com.* 1896-7. *Trans.* 2.
- 1894 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street.
- 1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Lib. Com.* 1895-6. *Trans.* 10.
- 1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.
- 1898 MORRISON, JAMES, M.D., St. Bartholomew's Hospital.
- 1885 MOTT, FREDERICK WALKER, M.D., F.R.S., Physician in charge of Out-patients, Charing Cross Hospital; Pathologist to the London County Council; 25, Nottingham place.
- 1896 MURPHY, JAMES KEOGH, M.A., M.B., B.C., 35, Prince's square, Bayswater.
- 1888 MURRAY, HUBERT MONTAGUE, M.D., Physician to Out-patients, and Lecturer on Pathology at, the Charing Cross Hospital; Physician to the Victoria Hospital for Children; 25, Manchester square.
- 1898 MURRAY, JOHN, 110, Harley street.
- 1880 MURRELL, WILLIAM, M.D., Physician to, and Lecturer on Materia Medica, Pharmacology, and Therapeutics at, the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

*Elected*

- 1892 MYDDELTON-GAVEY, E. HERBERT, 124, Harley street, Cavendish square, and 16, Broadwater Down, Tunbridge Wells.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.
- 1877 NETTLESHIP, EDWARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.
- 1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square. *Trans.* 1.
- 1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.
- 1891 OGLE, CYRIL, M.A., M.B., Assistant Physician to St. George's Hospital; 96, Gloucester place, Portman square.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 96, Gloucester place, Portman square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.
- 1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.
- 1896 OLIVER, GEORGE, M.D., 77, Wimpole street, Cavendish square, and Harrogate.
- 1892 OPENSHAW, T. HORROCKS, M.B., M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

*Elected*

- 1873 ORD, WILLIAM MILLER, M.D., Consulting Physician to St. Thomas's Hospital; 37, Upper Brook street, Grosvenorsquare. C. 1889-90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.
- 1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. C. 1897. *Lib. Com.* 1896—. *Trans.* 1.
- 1875 OSBORN, SAMUEL, 1A, Devonshire street, Portland place, and Maisonnnette, Datchet, Bucks.
- 1879 OWEN, EDMUND, M.B., Senior Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. C. 1896-7. *Trans.* 3.
- 1882 OWEN, ISAMBAARD, M.D., Deputy-Chancellor of the University of Wales; Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895—.
- 1892 PAGE, H. MARMADUKE, 26, Ashley gardens, Victoria street.
- 1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.
- 1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Foreign Associate of the Académie de Médecine, Paris; 5, Park square West, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

*Elected*

- 1886 **PAGET, STEPHEN**, Surgeon to the West London Hospital; Surgeon to the Throat and Ear Department of the Middlesex Hospital; 70, Harley street.
- 1895 **PARKER, CHARLES ARTHUR**, 41, Queen Anne street, Cavendish square.
- 1873 **PARKER, ROBERT WILLIAM**, Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. S. 1895-8. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87, 1892-5, 1898—. *Ho. Com.* 1892-5. *Trans.* 4.
- 1889 **PARSONS, J. INGLIS, M.D.**, Physician to the Chelsea Hospital for Women; 3, Queen street, Mayfair.
- 1883 **PASTEUR, WILLIAM, M.D.**, Senior Assistant Physician to the Middlesex Hospital; Consulting Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.
- 1891 **PATERSON, WILLIAM BROMFIELD**, 64, Brook street, Grosvenor square.
- 1891 **PATON, EDWARD PERCY, M.D., M.S.**, 84, Park street, Grosvenor square.
- 1865 **Pavy, FREDERICK WILLIAM, M.D., LL.D., F.R.S.**, Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.
- 1869 **PAYNE, JOSEPH FRANK, M.D.**, Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.
- 1894 **PEGLER, L. HEMINGTON, M.D.**, 27, Welbeck street.
- 1887 **PENROSE, FRANCIS GEORGE, M.D.**, Physician to St. George's Hospital and to the Hospital for Sick Children, Great Ormond street; 84, Wimpole street, Cavendish square. *Sci. Com.* 1889—.

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- 1897 PERRAM, CHARLES HERBERT, M.D., 22, Manchester square.
- 1890 PERRY, EDWIN COOPER, M.D., Physician to, and Demonstrator of Pathology at, Guy's Hospital; The Superintendent's House, Guy's Hospital.
- 1895 PHEAR, ARTHUR G., M.D., Assistant Physician and Pathologist to the Metropolitan Hospital, 13, Welbeck street, Cavendish square.
- 1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.
- 1884 PHILLIPS, GEORGE RICHARD TURNER, J.P., 28, Palace Court, Bayswater hill.
- 1888 PHILLIPS, JOHN, M.A., M.D., Obstetric Physician, King's College Hospital; Lecturer on Practical Obstetrics in King's College; Physician to the British Lying-in Hospital; 68, Brook street, Grosvenor square. *Trans.* 1.
- 1898 PHILLIPS, L. C. POWELL, St. Bartholomew's Hospital.
- 1889 PHILLIPS, SIDNEY, M.D., Physician and Lecturer on Medicine at St. Mary's Hospital; Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square. *Trans.* 1.
- 1867 PICK, THOMAS PICKERING, Consulting Surgeon to St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.
- 1884 PITT, GEORGE NEWTON, M.D., Physician to, and Pathologist at, Guy's Hospital; 15, Portland place. *Trans.* 1. *Referee*, 1897—.
- 1889 PITTS, BERNARD, M.A., M.C., Surgeon to St. Thomas's Hospital and Lecturer on Surgery; Surgeon to the Children's Hospital, Great Ormond street; 109, Harley street, Cavendish square. *Referee*, 1897—.

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- 1885 POLAND, JOHN, Surgeon to the City Orthopædic Hospital and Miller Hospital, Greenwich; 4, St. Thomas's street, London Bridge.
- 1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital; Consulting Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.
- 1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. V.P. 1896-7. *Referee*, 1872-81.
- 1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., 56, Park street, Grosvenor square.
- 1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence and Clinical Medicine in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road, and the Cheyne Hospital for Children, Chelsea; 32, Wimpole street, Cavendish square. C. 1890-91. *Referee*, 1887-9, 1892—. *Lib. Com.* 1895—. *Trans.* 2.
- 1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.
- 1867 POWELL, SIR RICHARD DOUGLAS, Bart., M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton, and to the Ventnor and Dental Hospitals; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee*, 1879-83, 1886. *Trans.* 3.
- 1887 POWER, D'ARCY, M.A., M.B., Assistant Surgeon at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 2.

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- 1867 **Power, HENRY**, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891-2. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.
- 1857 **Priestley, SIR WILLIAM OVEREND**, M.D., LL.D., M.P., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.
- 1883 **PRINGLE, JOHN JAMES**, M.B., C.M., Physician in Charge of Skin Department at the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.
- 1874 **PURVES, WILLIAM LAIDLAW**, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.
- 1877 **PYE-SMITH, PHILIP HENRY**, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893-4. *Lib. Com.* 1887-93. *Referee*, 1897—. *Trans.* 1.
- 1898 **RAMSAY, HERBERT MURRAY**, 35A, Hertford street.
- 1893 **RANKIN, GUTHRIE**, 4, Chesham street, Belgrave square.
- 1892 **RAYNER, HENRY**, M.D., 2, Harley street, Cavendish square.
- 1869 **READ, THOMAS LAURENCE**, 11, Petersham terrace, Queen's gate.
- 1891 **REECE, RICHARD JAMES**, 62, Addison gardens.
- 1891 **RENDEL, ARTHUR BOWEN**, M.A., M.B., B.C., 43, Albion street, Hyde Park.
- 1887 **RICHARDSON, GILBERT**, M.A., M.D., Hillside, Putney hill.
- 1863 **RINGER, SYDNEY**, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889-97. *Trans.* 6.

*Elected*

- 1896 ROBERTS, CHARLES HUBERT, M.D., 21, Welbeck street.
- 1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.
- 1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Medicine, and of Clinical Medicine, in University College, London; Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894-5. *Sci. Com.* 1889—.
- 1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Referee*, 1897—. *Trans.* 2.
- 1898 ROBERTSON, F. W., 54, Bernard street, Russell square.
- 1896 ROBINSON, HENRY BETHAM, M.S., Assistant Surgeon to, and Surgeon in Charge of the Throat Department, St. Thomas's Hospital; Assistant Surgeon to the East-London Hospital for Children, Shadwell; 1, Upper Wimpole street.
- 1890 ROLLESTON, HUMPHRY DAVY, M.D., Senior Assistant Physician to, and Lecturer on Pathology at, St. George's Hospital; Senior Physician to Out-patients, Victoria Hospital for Children; 112, Harley street, Cavendish square.
- 1857 ROSE, HENRY COOPER, M.D., Consulting Medical Officer to the Hampstead Dispensary and to the Soldiers' Daughters' Home, Hampstead; 16, Warwick road, Maida Vale. C. 1886-7. *Trans.* 1.
- 1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery in King's College; Senior Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.
- 1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Surgeon to the Royal Free Hospital; 38, Queen Anne street. *Trans.* 1.



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- 1882 **ROUTH, AMAND JULES McCONNEL, M.D., B.S.,** Obstetric Physician, and Lecturer on Midwifery, at the Charing Cross Hospital; Physician to the Samaritan Free Hospital for Women and Children; 14A, Manchester square.
- 1849 **Routh, CHARLES HENRY FELIX, M.D.,** Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.
- 1891 **RUSSELL, J. S. RISIEN, M.D.,** Assistant Physician to University College Hospital, and Pathologist to the National Hospital for the Paralysed and Epileptic, Queen square; 4, Queen Anne street, Cavendish square. *Trans.* 1.
- 1886 **SAINSBURY, HARRINGTON, M.D.,** Physician to the Royal Free Hospital; Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.
- 1869 **Sansom, ARTHUR ERNEST, M.D.,** Physician to the London Hospital; Consulting Physician, North - Eastern Hospital for Children; 84, Harley street, Cavendish square. *C.* 1887-8. *Referee*, 1889—. *Trans.* 3.
- 1845 **Saunders, SIR EDWIN,** Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; Fairlawn, Wimbledon Common. *C.* 1872-3.
- 1879 **SAVAGE, GEORGE HENRY, M.D.,** Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square. *C.* 1898—.
- 1883 **SCHÄFER, EDWARD ALBERT, LL.D., F.R.S.,** Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.
- 1892 **SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.P.H.,** Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.

*Elected*

- 1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, North Kensington.
- 1863 **Sedgwick**, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.
- 1892 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook street, Hanover square.
- 1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.
- 1877 SEMON, SIR FELIX, M.D., Physician for Diseases of the Throat to the National Hospital for Epilepsy and Paralysis, Queen square; 39, Wimpole street, Cavendish square. C. 1895-7. *Lib. Com.* 1894-5. *Trans.* 3.
- 1894 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.
- 1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square. *Referee*, 1897—. *Trans.* 2.
- 1886 SHAW, LAURISTON ELGIE, M.D., Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.
- 1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Referee*, 1897—. *Trans.* 6.
- 1896 SHORE, THOMAS WILLIAM, M.D., Heathfield, Alleyn park, Dulwich.
- 1893 SIBLEY, WALTER KNOWSLEY, M.D., B.C., Senior Physician to Out-patients, North-West London Hospital; 1, Duke street mansions, Grosvenor square.
- 1848 **Sieveking**, SIR EDWARD HENRY, M.D., LL.D., F.S.A. Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

*Elected*

- 1886 **SILCOCK, ARTHUR QUARRY, B.S.**, Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.
- 1842 **SIMON, SIR JOHN, K.C.B., F.R.S., Hon. M.D.** Dublin, 1887, Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee*, 1851-3, 1866-81. *Trans.* 1.
- 1892 **SIMS, FRANCIS MANLEY BOLDERO**, 12, Hertford street, Mayfair.
- 1893 **SISLEY, RICHARD, M.D.**, 1, Park row.
- 1894 **SLATER, CHARLES, M.B.**, 81, St. Ermin's mansions, Westminster.
- 1896 **SLOANE, JOHN STRETTON, M.B., B.S., B.Sc.**, 3, Montagu mansions, Portman square.
- 1890 **SMALE, MORTON**, 22A, Cavendish square.
- 1879 **SMITH, E. NOBLE**, Surgeon to the City Orthopædic Hospital; Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.
- 1881 **SMITH, EUSTACE, M.D.**, Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.
- 1891 **SMITH, G. COCKBURN, M.D.**, 5, Inverness gardens, Kensington.
- 1838 **SMITH, HENRY SPENCER**, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.
- 1866 **SMITH, HEYWOOD, M.A., M.D.**, 18, Harley street, Cavendish square.

*Elected*

- 1889 SMITH, ROBERT PERCY, M.D., B.S., 36, Queen Anne street.
- 1892 SMITH, SOLOMON CHARLES, M.D., Consulting Surgeon to the Royal Halifax Infirmary, and Physician to the Westminster General Dispensary; Four Oaks, Walton-on-Thames.
- 1863 Smith, SIR THOMAS, Bart., Surgeon Extraordinary to H.M. the Queen; Consulting Surgeon to St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.
- 1874 Smith, WILLIAM ROBERT, M.D., D.Sc., F.R.S. Edin., Barrister-at-Law, Professor of Forensic Medicine, and Director of the Laboratories of State Medicine in King's College, London; Medical Officer to the School Board for London; 74, Great Russell street. *Trans.* 1.
- 1865 Southey, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.
- 1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 104, Harley street. *Referee*, 1894—.
- 1887 SPENCER, WALTER GEORGE, M.B., M.S., Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.
- 1888 SPICER, ROBERT HENRY SCANES, M.D., Surgeon to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.
- 1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.
- 1875 SPITTA, EDMUND JOHNSON, Ivy House, 31, South Side, Clapham Common, Surrey.

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- 1851 **Spitta**, ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.
- 1885 **SQUIRE**, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 2.
- 1897 **STAINER**, EDWARD, M.A., M.B., 43, Vincent square.
- 1856 **Stocker**, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1884 **STONHAM**, CHARLES, Surgeon to, and Lecturer on Surgery and Teacher of Operative Surgery at, the Westminster Hospital; Surgeon to the Poplar Hospital for Accidents; 4, Harley street, Cavendish square.
- 1896 **SUTHERLAND**, GEORGE ALEXANDER, M.D., Physician to Paddington Green Children's Hospital; Assistant Physician to the North-West London Hospital; 9, Old Cavendish street.
- 1871 **Sutherland**, HENRY, M.D., Physician to Newlands House and Otto House Private Asylums, and to the St. George's Dispensary; 21, New Cavendish street.
- 1883 **SUTTON**, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; Surgeon to the Chelsea Hospital for Women; 48, Queen Anne street, Cavendish square. *Trans.* 6.
- 1896 **SWAN**, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 4, Devonport street, Hyde Park.
- 1890 **SYERS**, HENRY WALTER, M.D., 40, Wimpole street.
- 1886 **SYMONDS**, CHARTERS JAMES, M.S., M.D., Surgeon to, and in charge of the Throat Department at, Guy's Hospital; 58, Portland place.
- 1875 **TAY**, WARREN, Senior Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, and to the Hospital for Diseases of the Skin, Blackfriars; Consulting Surgeon to the North-Eastern Hospital for Children; 4, Finsbury square.

*Elected*

- 1873 TAYLOR, FREDERICK, M.D., *Trustee*; Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-6. *Sci. Com.* 1889—. *Referee*, 1887-8. *Trans.* 3.
- 1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; Physician to the North-Eastern Hospital for Children, and to the National Orthopædic Hospital; 49, Welbeck street, Cavendish square. *Trans.* 1.
- 1890 TAYLOR, SEYMOUR, M.D., Assistant Physician, West London Hospital; 16, Seymour street, Portman square.
- 1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.
- 1874 THIN, GEORGE, M.D., 63, Harley street, Cavendish square. C. 1893-4. *Trans.* 14.
- 1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee*, 1876-7. *Trans.* 1.
- 1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the Société de Chirurgie, Paris; 35, Wimpole street, Cavendish square. V.P. 1888. C. 1869. *Trans.* 8.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.
- 1892 THOMSON, STCLAIR, M.D., Physician to the Throat Hospital, Golden Square; Surgeon to the Royal Ear Hospital, London; 28, Queen Anne street, Cavendish square. *Trans.* 1.

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- 1892 THORNE, WILLIAM BEZLY, M.D., 53, Upper Brook street.
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women, and to the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893-95. *Trans.* 5.
- 1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.
- 1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 9, Park crescent, Portland place. C. 1887. V.P. 1897—. *Lib. Com.* 1879.
- 1882 TOOTH, HOWARD HENRY, M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square; Assistant Physician, late Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; 34, Harley street, Cavendish square. *Sci. Com.* 1896—.
- 1879 TREVES, FREDERICK, Surgeon-in-Ordinary to H.R.H. the Duke of York; Surgeon to the London Hospital; 6, Wimpole street, Cavendish square. C. 1895-6. *Referee*, 1890-5. *Sci. Com.* 1889-95. *Trans.* 5.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1897 TUNNICLIFFE, FRANCIS WHITTAKER, M.D., 6, Devonshire street.
- 1889 TURNBULL, GEORGE LINDSAY, M.D., Grove House, 76, Ladbroke grove.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.D., Physician to the London Hospital; Consulting Physician to the North-Eastern Hospital for Children; 15, Finsbury square. C. 1895-7.

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- 1882 TURNER, GEORGE ROBERTSON, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Visiting Surgeon to the Seamen's Hospital, Greenwich; 49, Green street, Park lane.
- 1896 TURNER, WILLIAM ALDREN, M.D., 13, Queen Anne street, Cavendish square.
- 1896 TURNEY, HORACE GEORGE, M.D., 68, Portland place.
- 1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and Surgeon to the Royal London Ophthalmic Hospital; 100, Harley street, Cavendish square.
- 1876 VENN, ALBERT JOHN, M.D., 70A, Grosvenor street.
- 1870 VENNING, EDGCOMBE, 30, Cadogan place. C. 1898—.
- 1865 VERNON, BOWATER JOHN, Senior Ophthalmic Surgeon to St. Bartholomew's Hospital; 14, Clarges street, Piccadilly.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.
- 1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Assistant Physician to, Pathologist and Curator of the Museum, and Lecturer on Pathology at the Middlesex Hospital; Assistant Physician, Hospital for Sick Children, Great Ormond street; 31, Harley street.
- 1896 WAGGETT, ERNEST, M.B., B.C., Assistant Surgeon, London Throat Hospital; 45, Upper Brook street.
- 1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 47, Weymouth street, Portland place.
- 1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.
- 1896 WALDO, FREDERICK JOSEPH, M.D., 1, Plowden Buildings, Temple.



*Elected*

- 1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.
- 1888 WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to the Charing Cross Hospital; 26, Welbeck street, Cavendish square.
- 1896 WALSHAM, HUGH, M.A., M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest; Assistant Medical Officer in Electrical Department, St. Bartholomew's Hospital; 114, Harley street, Cavendish square.
- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Hospital, the Bromley Cottage Hospital, and the Hospital for Hip Disease, Sevenoaks; 77, Harley street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 7.
- 1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.
- 1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 31, Grosvenor street.
- 1894 WARD-HUMPHREYS, GEORGE HERBERT, 9, Cavendish square.
- 1891 WARING, H. J., M.B., M.S., B.Sc., Surgical Registrar and Demonstrator of Operative Surgery, St. Bartholomew's Hospital; Surgeon, Metropolitan Hospital; 9, Upper Wimpole street.
- 1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace. *Trans.* 2.
- 1889 WASHBOURN, JOHN WICHENFORD, M.D., Physician to, Joint Lecturer on Physiology, and Lecturer on Bacteriology at, Guy's Hospital; Physician to the London Fever Hospital; 6, Cavendish place. *Trans.* 1.

*Elected*

- 1894 **WATERHOUSE, HERBERT FURNIVALL, C.M.**, Senior Assistant Surgeon and Lecturer on Anatomy, Charing Cross Hospital; Surgeon, Victoria Hospital for Children; 81, Wimpole street.
- 1861 **Watson, WILLIAM SPENCER, M.B.** (Lond.), late Surgeon to the Throat Department of the Great Northern Central Hospital; late Surgeon to the Royal South London Ophthalmic Hospital; 60, Queen Anne street. C. 1883-4. *Trans.* 1.
- 1879 **DE WATTEVILLE, ARMAND, M.D.**, 30, Welbeck street, Cavendish square.
- 1892 **WEAVER, FREDERICK POYNTON, M.D.**, Cedar Lawn, Hampstead Heath.
- 1891 **Weber, FREDERIC PARKES, M.D.**, Physician to the German Hospital, 19, Harley street.
- 1857 **WEBER, HERMANN, M.D.**, Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.
- 1896 **WEIR, ARTHUR NESHAM, M.B.**, 55, St. Charles square, Bayswater.
- 1895 **WELLS, SYDNEY RUSSELL, M.D.**, 24, Somerset street, Portman square.
- 1877 **WEST, SAMUEL, M.D.**, Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894-5. *Lib. Com.* 1892-4. *Trans.* 7.
- 1888 **WETHERED, FRANK JOSEPH, M.D.**, Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.
- 1881 **WHARRY, ROBERT, M.D.**, 6, Gordon square.
- 1875 **WHIPHAM, THOMAS TILLYER, M.D.**, Consulting Physician to St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

*Elected*

- 1891 **WHITE, CHARLES PERCIVAL, M.B., B.C.**, 144, Sloane street.
- 1881 **WHITE, WILLIAM HALE, M.D.**, Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888-97. *Trans.* 4.
- 1890 **WHITE-COOPER, W. G. O., M.B.**, 5, Courtfield road, Gloucester road.
- 1897 **WHITFIELD, ARTHUR, M.D.**, 12, Upper Berkeley street.
- 1877 **WHITMORE, WILLIAM TICKLE**, Consulting Surgeon to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.
- 1863 **Wilks, SIR SAMUEL, Bart., M.D., LL.D., F.R.S.**, Physician-Extraordinary to the Queen, Physician-in-Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; President of the Royal College of Physicians of London; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street. *Referee*, 1872-81.
- 1890 **WILLCOCKS, FREDERICK, M.D.**, Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.
- 1865 **Willett, ALFRED, Trustee**; Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892-8. *Trans.* 2.
- 1887 **WILLETT, EDGAR, M.B.**, 25, Welbeck street, Cavendish square.
- 1888 **WILLIAMS, CAMPBELL**, 18, Queen Anne street.
- 1866 **Williams, CHARLES THEODORE, M.A., M.D.**, *Trustee for Debenture-holders*; Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

*Elected*

- 1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children ; 101, Harley street. *Trans.* 1.
- 1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Duchess of York, Physician to H.R.H. the Princess Beatrice ; Emeritus Professor of Obstetric Medicine, University College, London ; Consulting Obstetric Physician to University College Hospital ; 63, Brook street, Grosvenor square. C.1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.
- 1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital ; Senior Physician to the North-Eastern Hospital for Children ; 29, Lower Seymour street, Portman square.
- 1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital ; 78, Harley street, Cavendish square.
- 1887 WOOD, THOMAS OUTTERSON, M.D., Senior Physician to the West End Hospital for Nervous Diseases ; 40, Margaret street, Cavendish square.
- 1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.
- 1892 WOODHEAD, GERMAN SIMS, M.D., Director of the Research Laboratory of R.C.P.Lond. and R.C.S.Eng. ; 1, Nightingale lane, Balham.
- 1890 WYNTER, WALTER ESSEX, M.D., Assistant Physician to the Middlesex Hospital ; 30, Upper Berkeley street, Portman square.

# LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

- |  |  |
|--|--|
| 1838 Henry Spencer Smith.                | 1859 Edward Tegart.                      |
| 1840 Sir James Paget, Bt., F.R.S.        | 1860 William Ogle, M.D.                  |
| 1842 Sir John Simon, K.C.B., F.R.S.      | Thomas Bryant.                           |
| 1845 Sir Edwin Saunders.                 | John Couper.                             |
| Edward U. Berry.                         | Henry Howard Hayward.                    |
| 1848 Sir Edward H. Sieveking, M.D.       | 1861 William Spencer Watson.             |
| John Clarke, M.D.                        | 1862 Lionel Smith Beale, M.B., F.R.S.    |
| 1849 C. H. F. Routh, M.D.                | Edmund Symes Thompson, M.D.              |
| 1851 John Birkett.                       | Reginald Edward Thompson, M.D.           |
| John A. Kingdon.                         | George Cowell.                           |
| Bernard E. Brodhurst.                    | 1863 Sir Samuel Wilks, Bt., M.D., F.R.S. |
| Robert J. Spitta, M.D.                   | Samuel Fenwick, M.D.                     |
| 1852 William Adams.                      | Julius Althaus, M.D.                     |
| Sir Henry Thompson.                      | Sydney Ringer, M.D., F.R.S.              |
| 1853 Robert Brudenell Carter.            | Sir Thomas Smith, Bart.                  |
| 1854 Sir Alfred B. Garrod, M.D., F.R.S.  | Arthur B. R. Myers.                      |
| 1855 William Marcet, M.D., F.R.S.        | William Sedgwick.                        |
| 1856 Charles J. Hare, M.D.               | 1864 John Harley, M.D.                   |
| William Bird.                            | Thomas William Nunn.                     |
| Jonathan Hutchinson, F.R.S.              | 1865 James Edward Pollock, M.D.          |
| Timothy Holmes.                          | Reginald Southey, M.D.                   |
| Alonzo H. Stocker, M.D.                  | George Fielding Blandford, M.D.          |
| 1857 Sir William Overend Priestley, M.D. | Sir Dyce Duckworth, M.D.                 |
| Hermann Weber, M.D.                      | Frederick W. Pavy, M.D., F.R.S.          |
| Henry Cooper Rose, M.D.                  | John Langton.                            |
| Henry Walter Kiallmark.                  | Frederick James Gant.                    |
| 1858 John William Ogle, M.D.             | Alfred Willett.                          |
| 1859 Wm. Howship Dickinson, M.D.         | Bowater John Vernon.                     |
| Edwin Thomas Truman.                     | Alfred Cooper.                           |
| Richard Barwell.                         | Christopher Heath.                       |

- 1866 Samuel Jones Gee, M.D.  
Charles Theodore Williams, M.D.  
Heywood Smith, M.D.  
William Selby Church, M.D.
- 1867 Achille Vintras, M.D.  
Sir R. Douglas-Powell, Bart., M.D.  
F. Howard Marsh.  
Henry Power.  
Sir William MacCormac, Bart.  
Thomas Pickering Pick.
- 1868 H. Charlton Bastian, M.D., F.R.S.  
Sir W. H. Broadbent, Bart., M.D.  
Thomas Buzzard, M.D.  
Walter Butler Cheadle, M.D.  
T. Henry Green, M.D.  
William Chapman Grigg, M.D.  
John Croft.  
George Eastes.
- 1869 Joseph Frank Payne, M.D.  
Arthur E. Sansom, M.D.  
Thomas Laurence Read.
- 1870 J. Warrington Haward.  
Edgcombe Venning.  
Clement Godson, M.D.  
Reginald Harrison.  
Robert Leamon Bowles, M.D.
- 1871 William Cayley, M.D.  
T. Lauder Brunton, M.D., F.R.S.  
J. Hughlings Jackson, M.D., F.R.S.  
Henry Sutherland, M.D.  
George Vivian Poore, M.D.  
Benjamin Duke, M.D.  
Philip Frank, M.D.
- 1872 T. Gilbert-Smith, M.D.  
George B. Brodie, M.D.  
Sir John Williams, Bart., M.D.  
Sir J. Fayrer, M.D., F.R.S.  
Charles S. Tomes, M.A., F.R.S.  
Sir William Bartlett Dalby.
- 1873 William Miller Ord, M.D.  
Frederick Taylor, M.D.  
Norman Moore, M.D.  
John Curnow, M.D.  
Sir William R. Gowers, M.D., F.R.S.  
Sir Wm. Guyer Hunter, M.D.  
Jeremiah McCarthy.  
Wm. Johnson Smith.  
Robert William Parker.  
Alex. O. MacKellar.  
Henry T. Butlin.  
Charles Higgens.  
William J. Walsham.
- 1874 Alfred Lewis Galabin, M.D.  
George Thin, M.D.
- 1874 John Mitchell Bruce, M.D.  
Henry Morris.  
William Laidlaw Purves.  
William Harrison Cripps.  
Henry G. Howse, M.S.  
Herbert William Page.  
Frederic Durham.  
William Robert Smith, M.D.
- 1875 Thomas T. Whipham, M.D.  
Francis Charlewood Turner, M.D.  
Thomas Crawford Hayes, M.D.  
Charles Henry Carter, M.D.  
Waren Tay.  
Edmund J. Spitta.  
Samuel C. Osborn.  
Fletcher Beach, M.B.
- 1876 Thomas Barlow, M.D.  
Wm. Lewis Dudley, M.D.  
Albert J. Venn, M.D.  
John Knowsley Thornton, M.B.  
N. Charles Macnamara.  
John N. C. Davies-Colley, M.C.
- 1877 Sir Felix Semon, M.D.  
Sidney Coupland, M.D.  
Francis Warner, M.D.  
William Ewart, M.D.  
Alfred Pearce Gould, M.S.  
Rickman J. Godlee, M.S.  
Alban H. G. Doran.  
George Ernest Herman, M.B.  
Samuel West, M.D.  
John Abercrombie, M.D.  
George Allan Heron, M.D.  
Joseph A. Ormerod, M.D.  
P. Henry Pye-Smith, M.D., F.R.S.  
Edward Nettleship.  
William Henry Bennett.  
William T. Whitmore.
- 1878 Sir Jas. Crichton-Browne, M.D.  
Fred. T. Roberts, M.D.  
Lord Lister, F.R.S.  
Clinton T. Dent.  
John H. Morgan.  
Donald W. Charles Hood, M.D.
- 1879 Edward Woakes, M.D.  
Armand de Watteville, M.D.  
Malcolm A. Morris.  
A. E. Cumberbatch.  
Edmund Owen.  
Arthur E. J. Barker.  
Frederick Treves.  
Thomas John MacLagan, M.D.  
Andrew Clark.  
Francis Henry Champneys, M.D.

- 1879 William Watson Cheyne, F.R.S.  
George Henry Savage, M.D.  
H. H. Clutton, M.A.  
Frederic S. Eve.  
E. Noble Smith.  
William Henry Allchin, M.D.  
F. G. Dawtrey Drewitt, M.D.
- 1880 Robert Alex. Gibbons, M.D.  
David Ferrier, M.D., F.R.S.  
Vincent Dormer Harris, M.D.  
Edmund Distin Maddick.  
Jas. John MacWhirter Dunbar, M.D.  
James William Browne, M.B.  
William Appleton Meredith, M.B.  
Malcolm Macdonald McHardy.  
A. Boyce Barrow.  
William Murrell, M.D.  
Leslie Ogilvie, M.B.  
George Ogilvie, M.B.  
Charles Edward Beevor, M.D.  
Thomas Colcott Fox, M.B.  
George Henry Makins.
- 1881 Francis de Havilland Hall, M.D.  
Robert Wharry, M.D.  
Cecil Yates Biss, M.D.  
Richard Clement Lucas, B.S.  
Stephen Mackenzie, M.D.  
William Hale White, M.D.  
Eustace Smith, M.D.  
Percy Kidd, M.D.  
Oswald A. Browne, M.D.  
W. Bruce Clarke, M.B.  
Dawson Williams, M.D.  
George Lindsay Johnson, M.D.  
Henry Edward Juler.  
Jonathan F. C. H. Macready.  
C. B. Lockwood.
- 1882 Philip J. Hensley, M.D.  
Ernest Clarke, M.D., B.S.  
John Barclay Scriven.  
George Robertson Turner.  
Howard Henry Tooth, M.D.  
Herbert Isambard Owen, M.D.  
Charles R. B. Keetley.  
Anthony A. Bowlby.  
Amand J. McC. Routh, M.D.  
Seymour J. Sharkey, M.D.  
William Lang.  
Henry Radcliffe Crocker, M.D.
- 1883 Edwin Clifford Beale, M.A., M.B.  
James Kingston Fowler, M.D.  
James Frederic Goodhart, M.D.  
John Charles Galton, M.A.  
W. Hamilton A. Jacobson, M.Ch.
- 1853 Walter H. Jessop, M.B.  
Walter Edmunds, M.C.  
Victor A. Horsley, F.R.S.  
Dudley Wilmot Buxton, M.D.  
Charles Douglas F. Phillips, M.D.  
John James Pringle, M.B.  
Henry Roxburgh Fuller, M.D.  
Wilmot Parker Herringham, M.D.  
Augustus Waller, M.D.  
William Pasteur, M.D.  
Edward Albert Schäfer, F.R.S.  
John Bland Sutton.  
William Rose, M.B.  
Storer Bennett.  
Robert Marcus Gunn, M.B.  
James Dixon Bradshaw, M.B.
- 1884 George Newton Pitt, M.D.  
Charles Stonham.  
Stanley Boyd, M.B.  
William Arbuthnot Lane, M.S.  
Arthur Marmaduke Sheild, M.B.  
Sidney Harris Cox Martin, M.D., F.R.S.  
George Lawson.  
Thomas Wakley, jun.  
F. Swinford Edwards.  
James Johnston, M.D.  
William Duncan, M.D.  
Charles Chinner Fuller.  
Jean Samuel Keser, M.D.  
George Richard Turner Phillips.  
Bilton Pollard.
- 1885 Alexander Haig, M.D.  
Theodore Dyke Acland, M.D.  
Frederick Walker Mott, M.D.  
James Berry.  
John Cahill, M.D.  
John Poland.  
Heinrich Port, M.D.  
A. C. Butler-Smythe.  
Charles Alfred Ballance, M.S.  
Walter S. A. Griffith, M.D.  
John Edward Squire, M.D.  
John D. Malcolm, M.B., C.M.  
Phineas S. Abraham, M.D.  
Henry Willingham Gell, M.B.
- 1886 Robert Maguire, M.D.  
Harrington Sainsbury, M.D.  
Cuthbert Hilton Golding-Bird, M.B.  
Benjamin Wainwright, M.B., C.M.  
Lauriston Elgie Shaw, M.D.  
Charters James Symonds, M.S.  
Robert Boxall, M.D.  
Allan Ogier Ward, M.D.

- 1886 Archibald Edward Garrod, M.D.  
Stephen Paget.  
William Radford Dakin, M.D.  
Samuel Herbert Habershon, M.D.  
Arthur Quarry Silcock.  
Arthur H. N. Lewers, M.D.
- 1887 Walter George Spencer.  
Thomas Outterson Wood, M.D.  
Edgar William Willett, M.B.  
Henry Lewis Jones, M.D.  
Francis George Penrose, M.D.  
Hugh Percy Dunn.  
Frederic William Hewitt, M.D.  
James Barry Ball, M.D.  
Gilbert Richardson, M.D.  
D'Arcy Power, M.B.  
Charles Arkle, M.D.  
John Gay.  
James Calvert, M.D.  
Percy J. F. Lush, M.B.
- 1888 Robert Henry Scanes Spicer, M.D.  
Jonathan Hutchinson, jun.  
Campbell Williams.  
James Donelan, M.B., C.M.  
John Anderson, M.D., C.I.E.  
Laurie Asher Lawrence.  
Arthur Pearson Luff, M.D., B.Sc.  
Albert Carless, M.S.  
Frederick C. Wallis, M.B., B.C.  
Charles James Cullingworth, M.D.  
Edmund Cautley, M.D., B.C.  
H. Montague Murray, M.D.  
Arthur Symons Eccles, M.B.  
Frank Joseph Wethered, M.D.  
Edmund Wilkinson Roughton, B.S.  
Frederick William Cock, M.D.  
John Phillips, M.D.
- 1889 Montagu Handfield-Jones, M.D.  
Norman M. MacLehose, M.B.  
David Henry Goodsall.  
Raymond Johnson, M.B.  
John Fletcher Little, M.B.  
Henry Work Dodd.  
George Lindsay Turnbull, M.D.  
Sir William Roberts, M.D., F.R.S.  
Sidney Phillips, M.D.  
William Charles Bull, M.B.  
George P. Field.  
John Wichenford Washbourn, M.D.  
Charles Henry Cosens.  
Henry Percy Dean, M.B., M.S.  
Alfred Samuel Gubb, M.D.  
William Hunter, M.D.  
J. Inglis Parsons, M.D.
- 1889 Bernard Pitts, M.B., M.C.  
Robert Percy Smith, M.D., B.S.  
Herbert R. Spencer, M.D., B.S.  
Nestor Isidore Chas. Tirard, M.I.
- 1890 John Rose Bradford, M.D., F.R.S.  
Roland Danvers Brinton, M.D.  
Charles D. B. Hale, M.D.  
Edwin Cooper Perry, M.D.  
Morton Smale.  
Frederick Willcocks, M.D.  
William T. Holmes Spicer, M.B.  
Thomas Henry Crowle.  
Henry Walter Syers, M.D.  
Seymour Taylor, M.D.  
William Alfred Wills, M.D.  
G. O. White-Cooper, M.B.  
Herbert William Allingham.  
William Anderson.  
William A. F. Bateman.  
James Jackson Clarke, M.B.  
Leonard G. Guthrie, M.D., B.Ch.  
G. William Hill, M.D., B.Sc.  
Edward Law, M.D., C.M.  
Patrick Manson, M.D., C.M.  
Humphry D. Rolleston, M.D.  
Arthur Henry Ward.  
Walter Essex Wynter, M.D., B.S.
- 1891 William Lee Dickinson, M.D.  
Herbert P. Hawkins, M.D., B.C.  
Cyril Ogle, M.A., M.B.  
Arthur F. Voelcker, M.D., B.S.  
Alfred Pownall Woodforde.  
Herbert T. Herring, M.B., B.S.  
Ernest Muirhead Little.  
Henry Charrington Martin, M.D.  
Frederick William Andrewes, M.I.  
Alfred Eddowes, M.D.  
Herbert Morley Fletcher, M.D.  
William Heaton Hamer, M.D.  
William Bromfield Paterson.  
Holburt Jacob Waring.  
Frederic Parkes Weber, M.D.  
F. E. Batten, M.D.  
Thomas Jessopp Bokenham.  
Norman Dalton, M.D.  
P. R. W. De Santi.  
P. W. Dove.  
William J. Gow, M.D.  
Charles Arthur Mercier, M.B.  
Paul Frank Moline, M.B.  
Edward Percy Paton, M.D.  
Arthur Bowen Rendel, M.B., B.C.  
James Samuel Risien Russell, M.I.  
George Cockburn Smith, M.D.



- 1891 Charles Percival White, M.B., B.C. W. Page May, M.D.  
Richard J. Reece.
- 1892 J. Dundas Grant, M.D.  
R. J. Bliss Howard, M.D.  
Thomas Horrocks Openshaw, M.B.  
William Bezly Thorne, M.D.  
German Sims Woodhead, M.D.  
W. H. Russell Forsbrook, M.D.  
John Harold.  
William Ward Leadam, M.D.  
John Alfred Masters, M.D.  
Gustave Schorstein, M.B.  
Charles Sempill de Segundo.  
John Tweedy.  
E. H. Myddelton-Gavey.  
E. Matthews James.  
J. S. Selwyn-Harvey, M.D.  
StClair Thomson, M.D.  
F. Manley B. Sims.  
F. Poynton Weaver, M.D.  
Henry Rayner, M.D.  
Walter S. Lazarus-Barlow, M.D.  
H. Marmaduke Page.
- 1893 James Taylor, M.D.  
Howard Barrett.  
Robert Cozens Bailey, M.B.  
Henry Albert Caley, M.D.  
Arthur Edward Giles, M.D.  
Miles Miley, M.B.  
D. Watkin Roberts, M.D.  
Leonard A. Bidwell.  
Frédéric F. Burghard, M.D., M.S.  
J. H. Drysdale, M.B.  
William McAdam Eccles, M.S.  
Vaughan Harley, M.D.  
George Herschell, M.D.  
Arnold Lawson.  
Guthrie Rankin.  
Walter Knowsley Sibley, M.D.  
Richard Sisley, M.D.
- 1894 Richard Gill.  
Joseph Sefton Sewill.  
Thomas Vincent Dickinson, M.D.  
Herbert Edward Durlham, M.B.  
Alexander Morison, M.D.  
L. Hemington Pegler, M.D.  
Herbt. Furnivall Waterhouse, C.M.  
Percy Furnivall.  
R. L. Langdon-Down, M.B., B.C.  
Allan Macfadyen, M.D., B.S.  
Ernst Michels, M.D.  
Wm. Rivers Pollock, M.B., B.C.  
Charles Slater, M.B.
- 1895 Charles Arthur Parker.  
Sydney Russell Wells, M.D.  
Alfred Milne Gossage, M.B.  
Robert Murray Leslie, M.B.  
James Galloway, M.D.  
David Bridge Lees, M.D.  
Arthur G. Phear, M.D.
- 1896 Joseph Lockhart Downes, M.B.  
Edward Wilberforce Goodall, M.D.  
James Ernest Lane.  
George Oliver, M.D.  
George Alex. Sutherland, M.D.  
Charles Buttar, M.D.  
P. J. Freyer, M.D., I.M.S., M.A.  
Percival Horton-Smith, M.D.  
Frederick Henry Lewis, M.B.  
James Keogh Murphy, M.B.  
Thomas William Shore, M.D.  
John Stretton Sleane, M.B.  
William Aldren Turner, M.D.  
Arthur Nesham Weir, M.B.  
John Brian Christopherson, M.B.  
Charles Hubert Roberts, M.D.  
John W. Watson Stephens, M.B.  
Charles R. J. Atkin Swan, M.B.  
James Kingston Barton.  
J. Walter Carr, M.D.  
John H. Dauber, M.A., M.B., B.Ch.  
Alexander Grant Russell Foulerton.  
L. Vernon Jones, B.A., M.D., B.Ch.  
Alexander MacGregor, M.D.  
Henry Betham Robinson.  
Horace George Turney, M.D.  
Ernest Waggett, M.B., B.C.  
Frederick Joseph Waldo, M.D.  
Hugh Walsham, M.B.
- 1897 Comyns Berkeley, M.B., B.C.  
William Arthur Brailey, M.D.  
James Cantlie, M.B.  
Raymond H. Payne Crawford, M.D.  
Louis Jenner, M.B.  
W. J. Collings Merry, M.D., B.Ch.  
Charles Herbert Perram, M.D.  
Francis Whittaker Tunnicliffe, M.D.  
Arthur Whitfield, M.D.  
Arthur A. Jamison, M.D.  
Edward Stainer, M.A., M.B.  
Alfred G. Levy, M.D.  
A. P. Beddard, M.B.  
G. F. Blacker, M.D.  
W. S. Colman, M.D.  
W. Gladstone Clark.  
F. W. Goodbody, M.D.  
R. Hutchison, M.D.

- |   |  |
|---|--|
| <p>1897 Harold Low.<br/>         1898 J. H. Bryant, M.D.<br/>             W. H. Corfield, M.D.<br/>             L. A. Dunn, M.S.<br/>             E. Hurry Fenwick.<br/>             A. Downing Fripp, M.S.<br/>             A. Corrie Keep, M.D.<br/>             A. C. Latham, M.D.<br/>             J. B. Lawford.<br/>             John McFadyean.<br/>             H. Murray Ramsay.<br/>             J. F. H. Broadbent, M.D.<br/>             H. Ronald Carter.<br/>             A. Stark Currie, M.D.</p> | <p>1898 P. J. Edmunds.<br/>             Alexander Granville.<br/>             James Morrison, M.D.<br/>             J. S. Edkins.<br/>             T. Jeeves Horder.<br/>             L. C. Powell Phillips.<br/>             F. W. Robertson.<br/>             S. Jervois Aarons, M.D.<br/>             Willmott Evans.<br/>             John Murray.<br/>             W. Adams Frost.<br/>             S. Backhouse Hulke.<br/>             C. R. C. Lyster.</p> |
|---|--|

*Elected*

- 1886 *BANKS, WILLIAM MITCHELL*, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.
- 1882 *BARKER, FREDERICK CHARLES*, M.D., Surgeon-Major, Bombay Medical Service.
- 1881 *BARNES, HENRY*, M.D., LL.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.
- 1861 *BARNES, ROBERT*, M.D., Lias, Hants. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.
- 1840 *BARROW, BENJAMIN*, Consulting Surgeon to the Royal Isle of Wight Infirmary; St. John's Lodge, Ryde.
- 1860 *Bealey, ADAM*, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.
- 1856 *BEARDSLEY, AMOS*, F.L.S., The Towers, Grange-over-Sands, Lancashire.
- 1896 *Belben, FRANK*, M.B., Royal Infirmary, Hull.
- 1880 *BENNETT, ALEXANDER HUGHES*, M.D. (Travelling).
- 1889 *BENTLEY, ARTHUR J. M.*, M.D., Mena House, Pyramids, Cairo, Egypt.
- 1872 *BEVERLEY, MICHAEL*, M.D., Consulting Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.
- 1865 *Bickersteth, EDWARD ROBERT*, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.
- 1892 *BICKERSTETH, ROBERT ALEXANDER*, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary: 2, Rodney street, Liverpool.
- 1849 *Birkett, EDMUND LLOYD*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.
- 1865 *BLANCHET, HILARION*, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.

*Elected*

- 1890 *BOSTOCK, R. ASHTON*, Surgeon, Scots Guards, Cefn Mor, Penmaen, Glamorganshire.
- 1869 *BOURNE, WALTER*, M.D. (Travelling).
- 1874 *BRADSHAW, A. F.*, C.B., Surgeon Major-General, 111, Banbury road, Oxford.
- 1876 *BRIDGES, ROBERT*, M.B., Manor House, Yattendon, Newbury, Berks.
- 1867 *BRIDGEWATER, THOMAS*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.
- 1891 *BRODIE, CHARLES GORDON*, Fernhill, Wootton Bridge, Isle of Wight.
- 1892 *BRONNER, ADOLPH*, M.D., Senior Surgeon to Bradford Eye and Ear Hospital; Laryngologist to Bradford Royal Infirmary; 33, Manor row, Bradford.
- 1894 *BROOK, WILLIAM HENRY BREFFIT*, M.D., B.S., 8, Eastgate, Lincoln.
- 1888 *BROWNE, HENRY LANGLEY*, Moor House, West Bromwich.
- 1881 *BROWNE, JOHN WALTON*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.
- 1864 *BUCKLE, FLEETWOOD*, M.D., Merton Lodge, Merton road, Southsea.
- 1851 *Cadge, WILLIAM*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich.  
*Trans.* 1.
- 1891 *CAMPBELL, HENRY JOHNSTONE*, M.D., 36, Manningham lane, Bradford.
- 1888 *CARTER, WILLIAM JEFFREYS BECHER*, Aliwal North, Cape Colony.
- 1868 *CAVAFFY, JOHN*, M.D., Physician to St. George's Hospital; C. 1887. *Referee*, 1896—. *Lib. Com.* 1888—. *Trans.* 1.
- 1884 *CHAFFEY, WAYLAND CHARLES*, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpelier road, Brighton.
- 1885 *CHAPMAN, PAUL MORGAN*, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford.  
*Trans.* 1.

*Elected*

- 1881 *Chavasse, THOMAS FREDERICK*, M.D., C.M., Senior Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital, the Corbett Hospital, Stourbridge, and Sutton Coldfield Dispensary; 22, Temple row, Birmingham. *Trans.* 3.
- 1873 *Chisholm, EDWIN*, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.
- 1892 *CLARK, JAMES CHARLES*, 35, Castle road, Bedford.
- 1897 *CLARK, W. GLADSTONE*, 1, North road, Surbiton.
- 1888 *CLARKE, ROBERT HENRY*, M.B., New House, Mersham, Ashford, Kent.
- 1857 *COATES, CHARLES*, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.
- 1868 *COCKLE, JOHN, A.M., M.D.*, Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.
- 1893 *COLE, ROBERT HENRY*, M.D., Moorcroft, Hillingdon, Uxbridge.
- 1891 *COOK, HERBERT GEORGE*, M.D., B.S., 22, Newport road, Cardiff.
- 1891 *COUMBE, JOHN BATTEN*, M.D., Christchurch street, Ipswich.
- 1869 *Cresswell, PEARSON R.*, C.B., Senior Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydfil.
- 1892 *CROSS, FRANCIS RICHARDSON*, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.
- 1895 *DARDEL, JEAN*, M.D., Aix-les-Bains, Savoy.
- 1879 *DARWIN, FRANCIS*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.
- 1874 *DAVIDSON, ALEXANDER*, M.D., Consulting Physician to the Liverpool Royal Infirmary; Emeritus Professor, University College, Liverpool; 2, Gambier terrace, Liverpool.

*Elected*

- 1878 *DAVY, RICHARD*, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.
- 1882 *DAWSON, YELVERTON*, M.D., Heathlands, Southbourne-on-Sea, Hants.
- 1889 *DELÉPINE, SHERIDAN*, B.Sc., M.B., C.M., Professor of Pathology, Owens College, Manchester. *Trans.* 1.
- 1867 *DRAGE, CHARLES*, M.D., Hatfield, Herts.
- 1884 *DRAGE, LOVELL*, M.D.Oxon., Burleigh Mead, Hatfield, Herts.
- 1885 *DRUMMOND, DAVID*, M.D., 7, Saville place, Newcastle-on-Tyne.
- 1880 *DRURY, CHARLES DENNIS HILL*, M.D., Bondgate, Darlington.
- 1871 *DUKES, CLEMENT*, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.
- 1867 *DUKES, MAJOR CHARLES*, M.D., Clarence Villa, Torrs park, Ilfracombe, North Devon.
- 1889 *DUNCAN, JOHN*, M.D., St. Petersburg, Russia.
- 1843 *DURRANT, CHRISTOPHER MERCER*, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.
- 1872 *EAGER, REGINALD*, M.D., Northwoods, near Bristol.
- 1887 *EASMON, JOHN FARRELL*, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.
- 1887 *ELLIOTT, JOHN*, 24, Nicholas street, Chester.
- 1848 *ELLIS, GEORGE VINER*, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.
- 1868 *ELLIS, JAMES*, M.D., The Sanatorium, Anaheim, Los Angeles County, California.
- 1889 *ELLISTON, WILLIAM ALFRED*, M.D., Stoke Hall, Ipswich.

*Elected*

- 1875 *Fagan, JOHN*, Consulting Surgeon to the Belfast Royal Hospital; 20, Fitzwilliam place, Dublin.
- 1897 *FAGGE, THOMAS HENRY*, M.D., Villa de la Porte Rouge, Monte Carlo.
- 1869 *FAIRBANK, FREDERICK ROYSTON*, M.D., Westcott, Dorking.
- 1872 *Fenwick, JOHN C. J.*, M.D., Physician to the Durham County Hospital; Long Framlington, Morpeth.
- 1879 *FINLAY, DAVID WHITE*, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician and Lecturer on Clinical Medicine to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891-3. *Trans.* 2.
- 1864 *Folker, WILLIAM HENRY*, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.
- 1896 *FORESTIER, HENRI*, M.D., Aix-les-Bains, Savoie, France.
- 1892 *FOSTER, MICHAEL GEORGE*, M.A., M.B., Villa Annita, San Remo.
- 1896 *FOULEBERTON, ALEXANDER GRANT RUSSELL*, Dunsdale, Mulgrave road, Sutton, Surrey.
- 1859 *FOX, EDWARD LONG*, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.
- 1884 *Franks, KENDAL*, M.D., Kilmurry, Hospital Hill, Johannesburg, South African Republic. *Trans.* 2.
- 1876 *FURNER, WILLOUGHBY*, M.D., Surgeon to the Sussex County Hospital; Brunswick square, Brighton.
- 1864 *Gairdner, SIR WILLIAM TENNANT*, M.D., K.C.B., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

*Elected*

- 1885 *GAMGEE, ARTHUR*, M.D., F.R.S., Emeritus Professor of Physiology in the Owens College, Victoria University, Manchester; Montreux, Switzerland.
- 1867 *GARLAND, EDWARD CHARLES*, Yeovil, Somerset.
- 1867 *GARLIKE, THOMAS W.*, Malvern Cottage, Churchfield road, Ealing.
- 1879 *GARSTANG, THOMAS WALTER HARROPP*, Headingley House, Knutsford, Cheshire.
- 1889 *Gaskell, WALTER HOLBROOK*, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; The Uplands, Great Shelford, Cambs.
- 1884 *GIBBES, HENEGAGE*, M.D., Health Officer, Detroit, Michigan, U.S.A.
- 1897 *GIBSON, GEORGE ALEXANDER*, M.D., D.Sc., 17, Alva Street, Edinburgh.
- 1897 *GILFORD, HASTINGS*, Norwood House, King's road, Reading. *Trans.* 1.
- 1893 *GORDON, WILLIAM*, M.B., M.C.
- 1890 *Gordon, WILLIAM*, M.D., Barnfield Lodge, Exeter.
- 1898 *GRAY, J. A.*, M.B., Wadham Lodge, Uxbridge road, Ealing.
- 1889 *GREENE, GEORGE EDWARD JOSEPH*, M.A., D.Sc., F.L.S., Monte Vista, Ferns, County Wexford.
- 1875 *Greenfield, WILLIAM SMITH*, M.D., Professor of Pathology and Clinical Medicine in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.
- 1882 *GRESSWELL, DAN ASTLEY*, M.A., M.D., D.P.H., Chairman, Board of Public Health, Melbourne, Victoria.
- 1889 *GRIFFITHS, JOSEPH*, M.A., M.D., C.M., Reader in Surgery in the University of Cambridge; Surgeon to Addenbrooke's Hospital; 63, Trumpington street, Cambridge. *Pro.* 1.
- 1870 *HAMILTON, ROBERT*, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magherabuoy, Portrush, Co. Antrim, Ireland.



*Elected*

- 1892 *HARSANT, WILLIAM HENRY*, The Tower House, Clifton road, Clifton, Bristol.
- 1854 *HAVILAND, ALFRED*, Ridgemount, Frimley Green, Surrey.
- 1890 *HAVILAND, FRANK PAPILLON*, M.D., B.C., 57, Warrior square, St. Leonard's-on-Sea.
- 1885 *HAWKINS, FRANCIS HENRY*, M.B., Physician to the Royal Berkshire Hospital; 73, London street, Reading. *Trans.* 1.
- 1861 *HAYWARD, WILLIAM HENRY*, Oxford road, Burnley, Lancashire.
- 1895 *HENDERSON, EDWARD ERSKINE*, B.A., M.B., B.C., [care of W. S. Henderson, Esq., 31, Lombard street, E.C.].
- 1843 *Holden, LUTHER*, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.
- 1894 *HOLLAND, JAMES FRANK*, M.D., St. Moritz, Engadine, Switzerland.
- 1868 *HOLLIS, WILLIAM AINSLIE*, M.D., Physician to the Sussex County Hospital; 1, Palmeira avenue, Hove. *Trans.* 1.
- 1846 *Holthouse, CARSTEN*, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.
- 1865 *HOWARD, BENJAMIN*, M.D. [New York, U.S.A.] *Trans.* 1.
- 1881 *HOWARD, HENRY*, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.
- 1896 *HUGHES, MATTHEW LOUIS*, Capt., Royal Army Medical Corps [care of Messrs. Holt and Co., 17, Whitehall place, S.W.]. *Trans.* 1.
- 1882 *HUMPHRY, LAURENCE*, M.D., 3, Trinity street, Cambridge.
- 1849 *HUSSEY, EDWARD LAW*, 24, Winchester road, Oxford. *Trans.* 1.
- 1896 *HYDE, SAMUEL*, M.D., Lismore House, 3, Hardwick street, Buxton.

*Elected*

- 1847 *IMAGE, WILLIAM EDMUND*, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.
- 1863 *JACKSON, THOMAS VINCENT*, Surgeon to the Wolverhampton and Staffordshire General Hospital, &c.; Whetstone House, Waterloo road south, Wolverhampton.
- 1883 *Jenkins, EDWARD JOHNSTONE*, M.D., The Australian Club, Sydney, New South Wales.
- 1851 *Jenner, SIR WILLIAM*, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; President of Royal College of Physicians, 1881-8; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.
- 1881 *JENNINGS, WILLIAM OSCAR*, M.D., 35, Rue Marbœuf, Avenue des Champs-Élysées, Paris.
- 1889 *JOHNSON, HAROLD J.*, Senior Assistant, Gloucester County Asylum, Gloucester.
- 1848 *JOHNSTONE, ATHOL ARCHIBALD WOOD*, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.
- 1876 *JONES, LESLIE HUDSON*, M.D., Limefield House, Cheetham hill, Manchester.
- 1875 *Jones, PHILIP SYDNEY*, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]
- 1865 *JORDAN, FURNEAUX*, Consulting Surgeon to the Queen's Hospital, Birmingham; Harborne, near Birmingham.

*Elected*

- 1893 *KANTHACK, ALFRED A.*, M.D., Professor of Pathology in the University of Cambridge ; Pathological Laboratory, New Museums, Cambridge.
- 1872 *KELLY, CHARLES*, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.
- 1848 *Kendell, DANIEL BURTON*, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.
- 1890 *Kerr, J. G. DOUGLAS*, M.B., C.M., 6, The Circus, Bath.
- 1877 *Khory, RUSTOMJEE NASERWANJEE*, M.D.Brux., Hormazd Villa, Khumballa hill, Bombay.
- 1898 *KLEFSTAD-SILLONVILLE, O.*, M.D., Aix-les-Bains, Savoie.
- 1888 *KINSEY, SIR WILLIAM RAYMOND*, C.M.G., Oriental Club, Hanover square. (Travelling.)
- 1889 *LANCASTER, ERNEST LE CRONIER*, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Hon. Physician to the Swansea and South Wales Institution for the Blind; Winchester House, Swansea, S. Wales.
- 1873 *Larcher, O.*, M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1862 *LATHAM, PETER WALLWORK*, M.D., Downing Professor of Medicine, Cambridge University, 1874-94; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1890 *Lawrie, EDWARD*, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.
- 1880 *LAYCOCK, GEORGE LOCKWOOD*, M.B., C.M., Melbourne, Victoria, Australia.
- 1886 *Lediard, HENRY AMBROSE*, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle.  
*Trans. 1.*

*Elected*

- 1882 *LEDWICH, EDWARD L'ESTRANGE*, Anatomist to the Royal College of Surgeons, Ireland; 30, Upper Fitzwilliam street, Dublin.
- 1895 *LEECH, DANIEL JOHN*, M.D., Elm House, Whalley Range, Manchester. *Sci. Com.* 1896—.
- 1883 *LEESON, JOHN RUDD*, M.D., C.M., Clifden House, Twickenham.
- 1869 *LEGG, JOHN WICKHAM*, M.D. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.
- 1898 *LINDSAY, JAMES*, M.A., M.D., 37, Victoria place, Belfast.
- 1872 *LITTLE, DAVID*, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.
- 1889 *Little, JAMES*, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens', and the Children's Hospitals; 14, Stephen's Green North, Dublin.
- 1871 *LITTLE, LOUIS STROMEYER*, Shanghai, China.
- 1894 *LOWE, THOMAS PAGAN*, 16, The Circus, Bath.
- 1867 *MABERLY, GEORGE FREDERICK*, Mailai Valley, Nelson, New Zealand.
- 1889 *MACALISTER, DONALD*, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Linacre Lecturer and Tutor, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.
- 1887 *MACDONALD, GEORGE CHILDS*, M.D.
- 1866 *MAGGOWAN, ALEXANDER THORBURN*, M.D., Vyvian House, Clifton park, Bristol.
- 1859 *M'Intyre, JOHN*, M.D., LL.D., Odiham, Hants.
- 1876 *MACKAY, EDWARD*, M.D., Physician to the Sussex County Hospital; Senior Physician to the Royal Alexandra Hospital for Sick Children; 56, Lansdowne place, Brighton.

*Elected*

- 1854 *Mackinder, DRAPER, M.D.*, 26, Denmark Villas, Hove, Sussex.
- 1893 *MACLEOD, SURGEON-COLONEL KENNETH, M.D.*, The Towers, Woolston, S. Hants.
- 1891 *MANBY, ALAN REEVE, M.D.*, Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales and to the Duke and Duchess of York at Sandringham; East Rudham, Norfolk.
- 1894 *MARRIOTT, CHARLES WILLIAM, M.D.*, Aubrey House, Bath road, Reading.
- 1892 *MARTIN, CHRISTOPHER, M.B., C.M.*, Surgeon to the Birmingham and Midland Hospital for Women; 35, George road, Edgbaston, Birmingham.
- 1883 *MAUDSLEY, HENRY CARR, M.D.*, 22, Collins street, Melbourne, Victoria.
- 1839 *MEADE, RICHARD HENRY*, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.
- 1895 *MILLS-ROBERTS, ROBERT HERBERT*, Hafod-ty, Llanberis, North Wales.
- 1887 *MIVART, FREDERICK ST. GEORGE, M.D.*, Local Government Board Inspector; 6, Edge hill, Wimbledon.
- 1896 *MOORE, JOHN WILLIAM, M.D., M.Ch.*, 40, Fitzwilliam square west, Dublin.
- 1891 *MORRIS, GRAHAM*, Wallington, Surrey.
- 1894 *MORSE, THOMAS HERBERT*, 10, Upper Surrey street, Norwich. *Trans.* 1.
- 1873 *MURRAY, J. IVOR, M.D., F.R.S.E.*, Granby House, Scarborough.
- 1881 *NALL, SAMUEL, M.B.*, Dryhurst Lodge, Disley, Stockport.
- 1889 *NAPIER, FRANCIS HORATIO, M.B.*, Cape Town.
- 1870 *NEILD, JAMES EDWARD, M.D.*, Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.
- 1895 *NEWSHOLME, ARTHUR, M.D.*, 11, Gloucester place, Brighton.

*Elected*

- 1868 *NICHOLLS, JAMES*, M.D., Trekenning House, St. Columb, Cornwall.
- 1849 *NORMAN, HENRY BURFORD*, The Manor-house, Drayton, Taunton, Somerset. *Lib. Com.* 1857.
- 1847 *Nourse, WILLIAM EDWARD CHARLES*, Ellesmere, Ilsham, Torquay (temporary).
- 1884 *OAKES, ARTHUR*, M.D., Narrabri, Cole Park road, Twickenham.
- 1880 *O'CONNOR, BERNARD*, A.B., M.D., Senior Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.
- 1896 *OGLE, JOHN GILBERT*, M.D., South Redlands, Reigate.
- 1855 *Ogle, WILLIAM*, M.A., M.D., Consulting Physician to the Royal Derbyshire Infirmary; The Elms, Duffield road, Derby.
- 1870 *OLDHAM, CHARLES FREDERIC*, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].
- 1883 *Oliver, THOMAS*, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.
- 1871 *O'Neill, WILLIAM*, M.D., C.M., late Physician to the Lincoln Lunatic Hospital, and Physician Lincoln General Dispensary, &c.; 2, Lindum road, Lincoln.
- 1890 *ORD, WILLIAM WALLIS*, M.D., The Hall, Salisbury.
- 1885 *ORMSBY, L. HEPENSTAL*, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.
- 1887 *PAGET, CHARLES EDWARD*, Medical Officer of Health to the County Council of Northamptonshire; County Hall, Northampton.
- 1858 *Paley, WILLIAM*, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.
- 1887 *PARDINGTON, GEORGE LUCAS*, M.D., 47, Mount Pleasant road, Tunbridge Wells.

*Elected*

- 1885 *PARKER, RUSHTON*, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.
- 1891 *PARKIN, ALFRED*, M.S., M.D., 24, Albion street, Hull. *Trans.* 1.
- 1879 *PEEL, ROBERT*, 120, Collins street east, Melbourne, Victoria.
- 1874 *PENHALL, JOHN THOMAS*, The Cedars, Broadwas-on-Teme, Worcester.
- 1879 *Pesikaka, HORMASJI DOSARHAI*, 43, Hornby road, Bombay.
- 1878 *Philipson, GEORGE HARE*, M.D., D.C.L., Professor of Medicine in Durham University; Consulting Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.
- 1891 *PIERCE, BEDFORD*, M.D., The Retreat, York.
- 1897 *PIGG, T. STRANGEWAYS*, 62, Jesus Lane, Cambridge.
- 1841 *Pitman, SIR HENRY ALFRED*, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.
- 1892 *POWELL, HERBERT ANDREWS*, M.A., M.D., M.Ch., Piccards Rough, Guildford.
- 1898 *PRENDERGAST, VINCENT*, 9, Rue Volney, Paris.
- 1897 *QUARTEY-PAPAFIO, BENJAMIN WILLIAM*, M.D., Accra, Gold Coast, West Africa.
- 1857 *VON RANKE, HENRY*, M.D., 3, Sophienstrasse, Munich.
- 1890 *RANSOM, WILLIAM BRAMWELL*, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.
- 1854 *RANSOM, WILLIAM HENRY*, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; 17, Park Valley, Nottingham. *Trans.* 1.
- 1882 *REID, SIR JAMES*, Bart., M.D., K.C.B., Resident Physician and Physician in Ordinary to H.M. the Queen, Windsor Castle.

*Elected*

- 1884 *REID, THOMAS WHITEHEAD*, M.D., Surgeon to the Kent and Canterbury Hospital ; St. George's House, Canterbury, Kent.
- 1881 *RICE, GEORGE*, M.B., C.M., Sutton, Surrey.
- 1889 *RIVERS, W. H. RIVERS*, M.D., St. John's College, Cambridge.
- 1871 *Roberts, DAVID LLOYD*, M.D., F.R.S.E., Consulting Obstetric Physician to the Manchester Royal Infirmary ; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester ; 11, St. John street, Manchester.
- 1889 *ROBERTS, LESLIE*, M.D., 46, Rodney street, Liverpool.
- 1873 *ROBERTSON, WILLIAM HENRY*, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital ; Buxton, Derbyshire.
- 1888 *Robinson, FREDERICK WILLIAM*, M.D., C.M., Huddersfield.
- 1889 *ROBSON, ARTHUR WILLIAM MAYO*, Professor of Surgery, Yorkshire College ; Senior Surgeon, Leeds General Infirmary ; 7, Park square, Leeds. *Trans.* 4. *Pro.* 1.
- 1885 *ROCKWOOD, WILLIAM GABRIEL*, M.D., Colombo, Ceylon.
- 1898 *ROGERS, LEONARD*, I.M.S. [care of Messrs. Watson & Co., Bombay]. *Trans.* 1.
- 1889 *ROSS, DANIEL MCCLURE*, M.D., Cedar Lodge, Littledown Road, Bournemouth.
- 1863 *ROWE, THOMAS SMITH*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary ; Union crescent, Margate, Kent.
- 1871 *RUTHERFORD, WILLIAM*, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh ; 14, Douglas crescent, Edinburgh.
- 1891 *RUFFER, MARC ARMAND*, M.D., The Quarantine Board, Alexandria.
- 1898 *SALTER, A.*, M.D., The Poplars, Sudbury, Harrow.
- 1855 *Sanderson, JOHN BURDON*, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford ; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.



*Elected*

- 1867 *SANDFORD, FOLLIOTT JAMES*, M.D., V.D., late Surgeon-Major, 2nd Batt. S.V.L. Infy., now Hon. Surgeon-Major; late Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.
- 1886 *SAUNDEY, ROBERT*, M.D., LL.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason University College; 83A, Edmund street, Birmingham.
- 1891 *SAUNDERS, FREDERICK WILLIAM*, M.B., B.C., Chieveley House, near Newbury, Berks.
- 1861 *SCOTT, WILLIAM*, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.
- 1897 *SEMPLE, EDWARD*, M.D., Grove house, Fenstanton, Hunts.
- 1897 *SEYMOUR, SURG.-MAJOR CHARLES*, Bareilly, North-West Provinces, India.
- 1887 *SIDEBOTHAM, EDWARD JOHN*, M.B., Erlesdene, Bowdon, Cheshire.
- 1857 *SIORDET, JAMES LEWIS*, M.B., Villa Cabrolles, Mentone, Alpes Maritimes, France.
- 1886 *SMITH, HOWARD LYON*, Buckland House, Buckland Newton, Dorchester.
- 1894 *SMITH, ROBERT SHINGLETON*, M.D., B.Sc., Deepholm, Clifton Park, Clifton, Bristol.
- 1894 *SMITH, THOMAS RUDOLPH*, M.B., B.C., 25, Bridge road, Stockton-on-Tees.
- 1868 *SOLLY, SAMUEL EDWIN*, Colorado Springs, Colorado, U.S.A.
- 1896 *STEPHENS, JOHN WILLIAM WATSON*, M.B., B.C., Pathological Laboratory, Cambridge.
- 1891 *STEVENS, SURG.-CAPT. CECIL ROBERT*, M.B., B.S., I.M.S., Eden Hospital, Calcutta.

*Elected*

- 1854 *STEVENS, HENRY*, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.
- 1884 *STEWART, EDWARD*, M.D., Brook House, East Grinstead.
- 1879 *Stirling, EDWARD CHARLES*, M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].
- 1865 *STOKES, SIR WILLIAM*, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.
- 1871 *STRONG, HENRY JOHN*, M.D., J.P., Hon. Consulting Physician, Royal Masonic Benevolent Institution, Croydon; Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.
- 1890 *Sympson, E. MANSEL*, M.D., B.C., Surgeon to the Lincoln County Hospital; Deloraine Court, Lincoln.
- 1870 *TAIT, LAWSON*, Surgeon to the Birmingham and Midland Hospital for Women; 195, Newhall street, Birmingham. *Trans.* 6.
- 1886 *TEALE, THOMAS PRIDGIN*, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.
- 1898 *THOMAS, J. LYNN*, Green Lawn, Pen-y-lan, Cardiff.
- 1890 *THOMAS, WILLIAM ROBERT*, M.D., Little Forest, Bath road, Bournemouth.
- 1891 *THOMSON, JOHN ROBERTS*, M.D., Monkchester, Bournemouth.
- 1883 *THURSFIELD, THOMAS WILLIAM*, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.
- 1880 *TIVY, WILLIAM JAMES*, 8, Lansdowne place, Clifton, Bristol.
- 1871 *Trend, THEOPHILUS W.*, M.D., 1, Grosvenor square, Southampton,

*Elected*

- 1881 *Treves, WILLIAM KNIGHT*, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.
- 1867 *TROTTER, JOHN WILLIAM*, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.
- 1873 *TURNER, GEORGE BROWN*, M.D., Camden House, Hemel Hempsted, Herts.
- 1894 *TURNER, PHILIP DYMCK*, M.D. (Travelling).
- 1891 *TWEED, REGINALD*, M.D., Higher Leyhill, Hambury Fort, Honiton.
- 1881 *TYSON, WILLIAM JOSEPH*, M.D., Senior Medical Officer of the Victoria Hospital, Folkestone; 10, Langhorne Gardens, Folkestone.
- 1854 *WADDINGTON, EDWARD*, Hamilton, Auckland, New Zealand.
- 1868 *Walker, ROBERT*, Clovelly, Bideford.
- 1887 *WALLACE, EDWARD JAMES*, M.D., Holmbush, Grove road, Southsea.
- 1867 *WALLIS, GEORGE*, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.
- 1883 *Walters, JAMES HOPKINS*, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.
- 1846 *WARE, JAMES THOMAS*, Tilford House, near Farnham, Surrey.
- 1861 *WATERS, A. T. HOUGHTON*, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.
- 1874 *WELLS, HARRY*, M.D., San Ysidro, Buenos Ayres, S. America.
- 1882 *WHARRY, CHARLES JOHN*, M.D., 14, Ewell road, Surbiton, Surrey.
- 1897 *WHITE, CHARLES POWELL*, General Hospital, Birmingham.

*Elected*

- 1881 *Whitehead, WALTER*, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; Professor of Clinical Surgery, Owens College, Victoria University; 499, Oxford road, Manchester. *Trans.* 1.
- 1885 *Whitla, WILLIAM*, M.A., M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; Consulting Physician to the Belfast Ophthalmic Hospital; 8, College square north, Belfast.
- 1852 *WIBLIN, JOHN*, The Hermitage, Clewer, Windsor. *Trans.* 1.
- 1870 *Wilkin, JOHN F.*, M.D., M.C., Rose Ash House, South Molton.
- 1883 *Willans, WILLIAM BLUNDELL*, Much Hadham, Herts.
- 1896 *WILLIAMS, ALFRED HENRY*, M.D., Rotorua, Harrow.
- 1859 *Williams, CHARLES*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.
- 1887 *WILSON, ARTHUR HERVEY*, M.D., 504, Broadway, Boston, U.S.A.
- 1889 *WISE, A. TUCKER*, M.D., Montreux, Switzerland.
- 1850 *Wise, ROBERT STANTON*, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.
- 1885 *WOLFENDEN, RICHARD NORRIS*, Rangemont, Seaford, Sussex.
- 1879 *WOODWARD, G. P. M.*, M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde Park, Sydney, New South Wales.
- 1892 *WRIGHT, ALMROTH EDWARD*, M.D., Ch.B., Oakhurst, Netley, Hants.

## ANNUAL MEETING.

*March 1st, 1898, at 5 p.m.*

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**WILLIAM HOWSHIP DICKINSON, M.D., President, in the Chair.**

**NORMAN MOORE, M.D.,** } **Hon. Secs.**  
**ROBERT WILLIAM PARKER,** }

The Minutes of the last Annual Meeting were read and confirmed.

The President nominated Dr. Eastes and Dr. Rolleston as Scrutineers of the Ballot, and declared the Ballot open until six o'clock.

Mr. R. W. Parker, Honorary Secretary, read the Report of the Council as follows :

### REPORT OF THE COUNCIL.

The Council have to report that the year 1897-98 has been fairly prosperous. Twenty-eight new Fellows have been elected, viz. 23 Resident, and 5 Non-resident. Since the last Annual Meeting the deaths of 5 Resident and 10 Non-resident Fellows have been reported. Two Fellows have resigned. The total Fellowship of the Society is thus :

Resident Fellows . . . . .	523
Non-resident Fellows . . . . .	271
Honorary Fellows (8 British, 16 Foreign)	24
<hr/>	
Total . . . . .	818

The revised Bye-laws, as sanctioned in the early part of the Session, have been found to work satisfactorily. Among the more important changes are the reduction of the radius for Resident Fellowship from 15 miles to 7 miles, and the establishment of fortnightly instead of monthly Council meetings.

One of the most important matters which has occupied the attention of the Council during the past year has been the extension of the hours during which the Library is open. Many of the Fellows are engaged at the hospitals during the afternoon, and it was thought that access to the Library during the forenoon would add to its usefulness. In future, therefore, the Library will be open from 11 a.m. to 6.30 p.m., and during these hours the Resident Librarian and the Sub-Librarian will be in attendance with the following exceptions, viz.: Thursday afternoons from 2 p.m., the Sub-Librarian; and on Saturday afternoons from 2 p.m. the Resident Librarian will be permitted to be absent. But on no occasion will the two be absent at the same time.

The papers submitted for reading and discussion have been of great value. That dealing with the prevention of enteric fever, the discussion on which occupied three evenings, and brought together a very large attendance of Fellows, deserves especial mention. Several distinguished authorities, not Fellows of the Society, took part in the discussion.

The Honorary Treasurers report as follows :

#### ASSETS AND LIABILITIES.

“ During the past year the financial position of the Society has improved, the amount of the Debenture debt having been reduced by £500 ; £300 having been paid off by the Society, and bonds for £200 having been most generously presented to the Society by Dr. Robert Barnes.

“The amount of unpaid accounts at the end of the year 1897 amounted to £924 11s. 6d., as against £1051 6s. 2d. at the end of the year 1896, a decrease of £126 14s. 8d.

#### RECEIPTS AND PAYMENTS.

“The total ‘receipts’ for the year as shown by the auditors’ statement appear to be £879 18s. 11d. less than in 1896. This is chiefly due to the disappearance from our accounts of the large balance which it used to be necessary to keep in hand on each 31st December, to meet the payment of the Debenture interest due on the following day.

“In the statement of receipts and payments for 1897, the two half-years’ interest on the Debentures appear within the financial year, and consequently the year began and ended with a smaller balance at the bankers.

“On the payment side of the statement there is an increase of £141 16s. 10d. in maintaining the premises and service of the Society, a large proportion of the increase being due to the rates and taxes having risen from £235 13s. 3d. to £286 18s. 2d.”

W. S. CHURCH,  
WARRINGTON HAWARD, } *Treasurers.*

The Honorary Librarians report as follows :

“We have pleasure in reporting that the past year has been one of steady progress.

“The additions to the Library during the past year number 527, exclusive of sets of books in continuation, periodicals, &c., the number of which is, of course, very large. £432 has been spent upon books and binding; the usual annual expenditure being £300.

“The donations to the Library have been numerous and valuable ; especial mention may be made of gifts of 25 volumes and nearly 200 pamphlets from Dr. Hare, and of 30 Russian pamphlets from Dr. Payne.

“The books issued during the past twelve months have numbered 3152 ; this does not include the issue of reference copies for a single night, nor of unbound periodicals and journals, which have a large circulation.

“The subscription account with Mr. Lewis has been well used, 207 volumes having been obtained on hire as against 170 last year. This plan works extremely well, and is a source of great satisfaction to Fellows in urgent need of books which have not been ordered for purchase.

“In July all books issued before 1897 were recalled, the borrowers being given the option of renewal if they desired to retain the books in their possession. The recall was carried out, it is believed, without any inconvenience to the Fellows, and was of great use in bringing back to the shelves many books that had probably been overlooked by their borrowers.

“An important event of the Library year is the recent order of the Council by which the library is now open at 11 a.m. instead of 1.30 p.m. as formerly. It is too soon yet to report upon the result of this change, but there is no doubt it will be greatly appreciated by the constantly increasing number of Fellows who use the Library.”

SAMUEL GEE, }  
R. J. GODLEE, } *Hon. Librarians.*

The Honorary Secretary (Mr. Pick) to the Committee appointed to investigate the subject of suspended anima-



tion in the drowned, reports that they are entering on a new arrangement, which they hope will facilitate their investigations, and enable them shortly to report to the Council.

The Honorary Secretaries (Drs. Archibald Garrod and Horton-Smith) report :

“ During the past year the Committee on Climatology and Balneology has made progress with the preparation of the second instalment of its report, and accounts of the climatology of several divisions of the country have been presented to and considered by the Committee. Dr. J. W. Moore, of Dublin, has been elected a member of the Committee, and has undertaken the Report on the Climates of Ireland.”

Dr. CHURCH, Honorary Treasurer, explained the audited Statement of Accounts, and answered questions which were raised in connection therewith.

On the motion of the PRESIDENT it was resolved—

(1) “ That the Report of the Council, together with the Treasurers’ audited Statement of Accounts, be adopted and printed in the next volume of the ‘ Transactions.’ ”

The PRESIDENT then delivered his

ANNUAL ADDRESS (p. xcix).

On the motion of Dr. CHURCH, seconded by Mr. BRYANT, it was resolved—

(2) “ That the best thanks of the Society be given to the retiring President, Dr. W. Howship Dickinson, for his valuable services to the Society during his term of office, and that he be requested to allow the address just delivered to be printed in the ‘ Transactions.’ ”

The PRESIDENT replied briefly.

On the motion of Dr. THEODORE WILLIAMS, seconded by Mr. D’ARCY POWER, it was resolved—

(3) “ That the best thanks of the Society be given to

the retiring Vice-Presidents, Dr. J. E. Pollock and Mr. Charles S. Tomes, for their valuable services to the Society during their respective terms of office."

On the motion of Sir THOMAS SMITH, seconded by Dr. NORMAN MOORE, it was resolved—

(4) "That the best thanks of the Society be given to the retiring Honorary Secretary, Mr. R. W. Parker, for his valuable services to the Society during his term of office."

Mr. PARKER briefly replied.

On the motion of Mr. JONATHAN HUTCHINSON, seconded by Dr. LEE DICKINSON, it was resolved—

(5) "That the best thanks of the Society be given to the retiring Members of Council, Dr. Abercrombie, Dr. Ferrier, Dr. Ormerod, and Dr. Gibbons, for their valuable services to the Society during their respective terms of office."

Dr. ABERCROMBIE (on behalf of the retiring Members of Council) briefly replied.

On the motion of Dr. HARE, seconded by Mr. ALFRED WILLETT, it was resolved—

(6) "That the Society desires to take this opportunity of placing on record its special and very hearty thanks to Mr. Holmes for his valuable services during a continuous period of about ten years, first as Chairman of the Building Committee, and next as Chairman of the House Committee. The meeting desires that a copy of this resolution shall be sent by the Honorary Secretaries to Mr. Holmes."

Mr. HOLMES acknowledged the vote of thanks.

The PRESIDENT declared the Ballot closed, and called for the Report of the Scrutineers. The Scrutineers reported that all the candidates nominated as Officers and Council for the ensuing year had been duly elected.

*President.*—Thomas Bryant.

*Vice - Presidents.*—George Fielding Blandford, M.D.; William Marcet, M.D., F.R.S.; Frederick James Gant; Reginald Harrison.

*Honorary Treasurers.*—William Selby Church, M.D. ; J. Warrington Haward.

*Honorary Secretaries.*—Norman Moore, M.D. ; Alfred Pearce Gould, M.S.

*Honorary Librarians.*—Samuel Jones Gee, M.D. ; Rickman J. Godlee, M.S.

*Members of Council.*—William Henry Allchin, M.D. ; Robert Leamon Bowles, M.D. ; John Mitchell Bruce, M.D. ; Francis Henry Champneys, M.D. ; George Henry Savage, M.D. ; William Watson Cheyne, F.R.S. ; Henry Hugh Clutton, M.B. ; Frederic S. Eve ; William Appleton Meredith, M.B., C.M. ; Edgcombe Venning.

The PRESIDENT then called upon Mr. Thomas Bryant to come to the platform, and invested him with the President's badge of office and master-key, and installed him in the Chair. The new President briefly addressed the meeting, and thanked the Fellows for his election.

# STATEMENT OF LIABILITIES AND ASSETS, 31st DECEMBER, 1897

LIABILITIES.		ASSETS.	
£	s. d.	£	s. d.
3 per Cent. First Mortgage Debentures . . .	*35,300 0 0	Freehold and Leasehold Property (As per Balance-sheet of 31st December, 1896).	51,150 0 0
Sundry Creditors . . . . .	924 11 6	Fixtures, Fittings, and Furniture Less 5 per cent. written off for depreciation . . . . .	1427 7 0 71 7 4
Balance, being surplus of Assets over Liabilities	25,679 11 9	Engravings (As per Balance-sheet of 31st December, 1896).	1355 19 8 555 0 0
<p>* This amount has been reduced by £500 since the previous statement, £300 of which has been paid in cash in respect to Debentures redeemed. The balance of £200 is a gift to the Society by Dr. Robert Barnes.</p>		Contents of Library as on December 31st, 1896 . . .	7693 0 0
		Added in 1897 . . . £455	
		Less Depreciation (50 per cent.) . . . . .	227
		Value of Gifts . . . . .	228 20
		Investment . . . . .	7941 0 0
		(New South Wales 4 per Cent. Inscribed Stock).	326 7 3
		Sundry Debtors for Rents . . . . .	448 11 9
		Cash at Bank . . . . .	127 4 7
			<u>£61,904 3 3</u>

NOTE.—The Society is also possessed of £656 16s. 11d. Consols, and £1 0s. 7d. in cash, but as the sum in question is held in trust for a specific purpose, viz. the Marshall Hall Memorial Prize Fund, the capital sum has not been included amongst the assets of the Society.

Audited and approved, **TOM MUNDY,**  
Chartered Accountant.  
15th February, 1898.

## STATEMENT OF RECEIPTS AND PAYMENTS FOR

		Receipts					
		£ s. d.		£ s. d.		£ s. d.	
Balance 1st January, 1897 :							
Cash in hand	.. ..			509 13 1			
Less overdraft at Bankers	.. ..			159 16 2			
				<hr/>		349 16 11	
<i>Subscriptions, Fees, &amp;c. :</i>							
426 Annual Subscriptions at £3 3s.	.. ..	1341 18 0					
60 Annual Subscriptions at £1 1s.	.. ..	63 0 0					
Composition Fees (Life)	.. ..	42 0 0					
Entrance Fees	.. ..	151 4 0					
		<hr/>		1598 2 0			
<i>Transactions and Proceedings :</i>							
Sold by Messrs. Longmans	.. ..	59 5 11					
„ Mr. H. K. Lewis	.. ..	1 11 2					
„ Resident Librarian	.. ..	12 13 2					
		<hr/>		73 10 3			
<i>Rents</i>	.. ..			2205 17 11			
<i>Interest :</i>							
On Permanent Endowment Fund	.. ..			12 12 4			
		<hr/>				3890 2 6	
<i>Debenture Account :</i>							
Amount overpaid, 31st December,							
1896	.. ..					41 18 11	
						<hr/>	
						<u>£4281 18 4</u>	

W. S. CHURCH,  
J. WARRINGTON HAWARD, } *Treasurers.*

15th February, 1898.

## THE YEAR ENDING 31ST DECEMBER, 1897

## Payments

	£	s.	d.	£	s.	d.
<i>Rent, Rates, &amp;c.</i> .. .. .				286	18	2
<i>Lighting, Cleaning, &amp;c.</i> .. .. .				333	6	1
<i>Repairs, Alterations, Furniture, &amp;c.</i> .. .. .				214	16	11
<i>Meeting Expenses</i> .. .. .				31	2	3
<i>Printing, Stationery, &amp;c.</i> .. .. .	141	3	0			
<i>Stamps and Telegrams</i> .. .. .	11	3	1			
				152	6	1
<i>Officers and Servants :</i>						
Salaries and Wages .. .. .				822	17	5
<i>Library</i> :—Books and Binding .. .. .				455	12	6
<i>' Transactions ' and ' Proceedings '</i> .. .. .				410	2	6
<i>Debentures :</i>						
Three Bonds redeemed .. .. .				300	0	0
Interest .. .. .				1034	6	8
<i>Legal Charges</i> .. .. .				25	17	8
<i>Jubilee Illumination</i> .. .. .	15	0	0			
<i>Less Amount received from Tenants</i> .. .. .	6	6	0			
				8	14	0
<i>Auditors' Fee</i> .. .. .				10	10	0
<i>" Spas " Committee: Copies of ' Climates and Baths ' to Contributors and Committee</i> .. .. .						
				11	9	6
<i>Miscellaneous</i> .. .. .				56	14	0
<i>Balance at Bank</i> .. .. .				127	4	7
				£4281	18	4

Audited and approved.

TOM MUNDY,  
Chartered Accountant.

## LIST OF PAPERS.

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N.B.—THE Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its *Transactions*.

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ADDRESS  
OF  
WILLIAM HOWSHIP DICKINSON, M.D.,  
PRESIDENT,  
AT THE  
ANNUAL MEETING, MARCH 1st, 1898.

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GENTLEMEN,—My first duty is to express my profound gratitude for the high honour you have conferred upon me in choosing me as your President. The uniform courtesy and consideration which I have received from the Fellows have made my duties in the Chair not only a pride but a pleasure, and I shall ever look back with un-mixed gratification upon the time when I was the head of this great Society.

Our prosperity during the last twelve months has been fully maintained ; twenty-eight new Fellows have been elected, which more than counterbalances numerically our losses by death and retirement, which together amount to sixteen. Papers have come in abundantly ; our discussions, whether prearranged or spontaneous, have been full of interest ; and I may say that in no respect has the work of the Society suffered deterioration. The recent prolongation of the Library hours fulfils a wish which has long been widely expressed, and cannot but add to the convenience of readers.

The Dinner, which was held on the 17th of last month, was attended by Fellows and guests to the number of eighty-eight, and proved, I believe, generally acceptable. Such gatherings cannot fail to increase the social feeling

which exists among the Fellows ; they may serve to introduce the Society to some who do not belong to it ; and they afford an opportunity of returning the hospitality of other associations. I should like to see these festivals annual, as matters of yearly routine rather than of uncertain and spasmodic recurrence.

We at this time lose the services of Mr. Parker, who has worked for the Society in several capacities. As Secretary he has been largely concerned in the laborious task of revising the Bye-Laws, and has been indefatigable in every matter which has fallen within his duties. Mr. Holmes also ceases to be our officer. As Chairman of the Building Committee, as President of the Society, and finally as Chairman of the House Committee, Mr. Holmes's services have been remarkable in length and continuity, and not less so for the devotion and energy with which they have been performed.

It now remains for me to read the names of those whose death we have to regret, and to notice in brief detail the lives of as many of them as time permits. I have written, however inadequately, of all whose death within the last twelve months has come to my knowledge, but time will not allow me to read more than a selection.

The death-roll, which includes fourteen names, is as follows :

Date of Election.	Name.			Date of Death.
1855	Pemberton, Oliver	...	...	March 7th, 1897.
1856	Hudson, C. E. Leopold B.	...	...	March 29th, 1897.
1862	Andrew, James	...	...	April 21st, 1897.
1871	Rivington, Walter	...	...	May 8th, 1897.
1885	Smith, James Greig	...	...	May 28th, 1897.
1859	Chance, Frank	...	...	July 1st, 1897.
1882	Roy, Charles Smart	...	...	October 4th, 1897.
1842	Fletcher, Thomas Bell Elcock	...	...	October 21st, 1897.
1839	Alcock, Sir Rutherford, K.C.B.	...	...	November 2nd, 1897.
1890	Cagney, James	...	...	November 17th, 1897.
1889	Freeman, Henry William	...	...	November 28th, 1897.
1883	Lund, Edward	...	...	February 4th, 1898.
1891	Remfry, Leonard	...	...	February 10th, 1898.
1878	Bindon, William John Vereker	...	...	

*Oliver Pemberton* was a conspicuous townsman of Birmingham, and a provincial surgeon of more than provincial repute. The details of his life and work have been fully chronicled in the medical and local papers, and little need be added to what has there found full expression. He was an essential item in the life of Birmingham, prominent as a surgeon, as a member of the Town Council, as a magistrate, and latterly as coroner. His striking appearance, his popular manners, his genial nature, the respect which he enjoyed, and the activity which brought him to the fore in each of the characters in which he acted on the stage of life, all these contributed to make him a person of note in the great town in which his lot was cast. But not only at Birmingham was he well known. He was an Honorary Fellow of the College of Surgeons of London, for a time he had a seat on the Council, and he was honoured with the diploma of Foreign Corresponding Secretary of the Society of Surgery in Paris. From the year 1852 until 1891 he was Surgeon to the General Hospital at Birmingham, and there lectured on surgery. He was the author of many writings on this subject, on Lithotomy in Children, on Excision of the Ankle-joint, and others, perhaps the most important of which was his work on the various forms of cancer. To our 'Transactions' he contributed one paper, which was on a case of Aneurismal Varix. His death occurred on March 7th, 1897, in his seventy-second year, as the result of malignant disease within the abdomen. Enough has been said to show that a noteworthy surgeon and a man who filled a large space in a great social centre has been removed from our list of Non-resident Fellows.

In *Charles Elliott Leopold Barton Hudson* we have to lament the loss of a man who might have attained to a distinguished position had he not been cut off in comparatively early life.

After much success as a student at the Middlesex Hospital, and holding with acceptance some of the junior offices there, he was in 1893 elected Assistant Surgeon to

the Hospital for Sick Children. In 1895 he became Assistant Surgeon to the Middlesex Hospital. He was well known and highly esteemed in connection with pathological work, of which he did much for his Hospital and for the Pathological Society. At that Society I used often to meet him, and always regarded him with liking and respect. He was at one time Secretary to the Committee on Swine Fever under the Board of Agriculture, and held the important Secretaryship of the Royal Commission on Tuberculosis. He was fitted for these responsible offices by his pathological knowledge and skill in investigation, by his industry, carefulness, and attention to minute details, and by his unfailing courtesy and urbanity.

Mr. Leopold Hudson died on the 29th of last March, in the thirty-fifth year of his age. The immediate cause of his death was cerebral apoplexy, which was sequent upon renal calculus. More regret naturally attaches to those who die young than to those who have had their full share of what this world has to offer. But we must more than commonly deplore the untimely fate of one to whom so much promise attached.

*Dr. James Andrew* was born at Whitby in the year 1829, the third and youngest son of the perpetual curate of that place. The three brothers all went to Oxford, took honours in classics, and became Fellows of their respective Colleges,—the eldest of Worcester, the second of Lincoln, the third, of whom I have to speak, of Wadham.

James Andrew took his degree in 1852. He was not elected Fellow until 1856, having passed the intervening years at Oxford as a coach. Upon receiving his Fellowship at the age of 27 he matriculated as a student of Medicine at the University of Edinburgh, and in the following year entered at St. Bartholomew's Hospital. Dr. Andrew was a development which is less frequent now than it once was—a physician grafted upon a classical or mathematical stock. Like Watson, Burrows, and the

late Dr. Henry Thompson, Dr. Andrew did not turn to medicine until he had spent some years in academical pursuits and attained a considerable age. All had attained high scholastic positions before descending to the grade of medical students. It may be doubted whether medicine is cultivated to the best advantage by those whose most acquisitive years have been spent in studies which have no direct relation to it, but the examples of Watson, Burrows, and Dr. Andrew himself suffice to show that some who enter the profession late may do well in it. Nevertheless in these latter days, when medicine has come to demand as preliminary to itself a scientific foundation which was unknown to our forefathers, and which requires education of hand as well as of brain, it may be thought well that this should be acquired before either body or mind has lost the adaptability of youth. Chemistry and physiology are the true antecedents of medicine, not Latin and Greek.

In 1861 Dr. Andrew became Demonstrator of Morbid Anatomy and Warden of the College of St. Bartholomew's. He now threw himself with devotion into practical pathology, and soon acquired some prominence at the Pathological Society. It was here that I first became acquainted with him. He was a frequent exhibitor, and was always listened to with respect. He was occasionally selected by the Society to report on specimens shown by others, and was a member of a Committee on Lardaceous Disease. In addition to his occupation among the dead, Dr. Andrew found employment among the living as Physician to the Royal General Dispensary of Bartholomew Close, and at the City of London Hospital for Diseases of the Chest. But the work of his life was not in societies or smaller institutions, but in the great hospital with which his memory will be chiefly associated. He became Assistant Physician in 1864, Joint Lecturer on Medicine in 1868, and full Physician in 1869. It is somewhat unusual to find a Lecturer on Medicine in a large school in the position of assistant physician, but

Dr. Andrew must have acquired considerable clinical experience in the offices he had previously held, and his lectures are described as full of instruction and of information which could not be obtained elsewhere. They were read from a carefully prepared manuscript, and were sound rather than brilliant. They required close attention, as his accumulated facts were seldom relieved by illustration.

Though he had a considerable private practice, it is within his own hospital, by his own pupils, and by his personal friends that he will be chiefly remembered. He was most esteemed by those who knew him best. He was an excellent clinical teacher, especially interesting and instructive on heart disease. The consideration and kindness with which he treated the patients in his wards presented, as I am assured, a noteworthy example.

At the College of Physicians he was prominent and influential. He held at different times the offices of Examiner, Councillor, Censor, Lumleian Lecturer, and Harveian Orator. For the Lumleian Lectures he took as his subject the *Ætiology of Phthisis*, for the Harveian Oration that of the regulation of the Pulmonary Circulation in relation to *Hæmoptysis* and the Action of the Vaso-motor Nerves. The estimation in which Dr. Andrew was held at the College was displayed by a tangible minority of votes at the Presidential election which resulted in the choice of Sir Andrew Clark. I recall an occasion in which his influence was manifested in a different manner. He once felt it his duty to protest against the selection of Fellows as nominated by the Council; the protest was endorsed by the College and the list withdrawn.

Dr. Andrew's amiability and benevolence had no admixture of weakness, he was even at times somewhat impetuous; he could take his line decidedly and hold it tenaciously. Some who knew him but superficially may have failed to discern the sterling qualities of courage,

straightforwardness, and integrity which lay beneath his unassuming and retiring demeanour. He was ever ready to extend his help, pecuniarily or otherwise, to those who sought it. He was careless of appearances, and conspicuously wanting in the art of self-advertisement. He was respected wherever known, but was less widely known than he deserved to be. The esteem in which he was held at his University was testified by his selection as Honorary Fellow of Wadham.

He possessed considerable literary power. Though not, as I should judge, a ready speaker, he could make a premeditated speech in graceful language and with force and effect. He was not a voluminous writer. Besides the lectures I have referred to he published an Inaugural Thesis on "Anasarca in Renal Disease" and two papers in the 'St. Bartholomew's Reports,' one on the "Systolic Apex Murmur," and the other on the kindred subject of the "Diseases of the Mitral Valve." He contributed a paper to the sixtieth volume of our 'Transactions,' in conjunction with Sir Dyce Duckworth, on a case of "Paralysis in a Child following Exposure to Heat."

He retired from the hospital in 1893 at the age of sixty-three. He had become bent, deaf, and prematurely old. He directed his course westwards, first to the New Forest, then to Bournemouth, and finally to Tavistock. He died on the 21st of last April at the age of sixty-seven. He had never quite recovered from an attack of influenza in the autumn of 1890, and remained subject to bronchitis, which was the chief cause of his death.

He presented the type or exemplar of the scholarly physician. He was possessed of learning, but not ostentatious in the display of it. He was well-informed in what was ascertained or accepted, thoughtful and judicious in the application of his knowledge. As a clinical teacher he was at his best, and is spoken of as no unworthy successor of the great physicians who preceded him in the same place. Absolutely honest, considerate in his dealings with others of the faculty, he in all



respects did honour to the great hospital to which he belonged.

*Walter Rivington* passed the greater part of his life in connection with the London Hospital, to which at his death he was Consulting Surgeon, having served his full time on the active staff, and held many offices in the school. He was a member of the Council of the College of Surgeons and of the Senate of the University of London. He obtained the latter position in competition with Sir Joseph, now Lord Lister. Mr. Rivington's views with regard to proposed changes in the University were more in accordance with those of the graduates than were those of the great surgeon and natural philosopher over whom he prevailed. Mr. Rivington, though distinguished as a writer on surgery, was perhaps less known as a practical surgeon than as a medical reformer. On the debatable ground of medical politics I am not qualified to enter. If personally I am more inclined to do honour to a surgeon for his achievements in the operating theatre than on the platform, I cannot forget that the politician, whose object it is to direct and control the efforts of others, has his use, though it is not always so obvious as is that of the physician or surgeon who brings his own brain or hand to bear upon the suffering body. We have some among us who are eminent in both capacities, in relation to the individual body and to the body politic.

Mr. Rivington was a man of high character and considerable gifts, mainly of a literary kind. He was brilliant as a student, and was doubly distinguished as a Gold Medallist at the University of London. I am informed and can readily believe that he was an excellent lecturer, whether on anatomy or surgery, and I have heard him speak with much force and readiness on matters in which he was interested. He was known as a writer on surgical subjects, and contributed five papers to our 'Transactions.' He was the author of a volume on 'Rupture of the Urinary Bladder' which is an exhaustive

compilation of the knowledge concerning it up to the year 1883, which was the date of its publication. This will probably long remain of use as a work of reference, though surgical practice has since progressed beyond the limits which are therein indicated. His most voluminous works were two essays, to each of which was awarded the Carmichael Prize by the College of Surgeons of Ireland. I have seen only the second, which was published in 1888. This is entitled 'The Medical Profession in the United Kingdom.' It begins with the ecclesiastical medicine of the Druids, and does not conclude until it has given due prominence to the medical ecclesiasticism of the Guild of St. Luke. There is much between the two, corporations, hospitals and dispensaries, the crimes of the resurrectionists and the virtues of the Medical Council.

I have said enough to declare the abilities of Mr. Rivington, which were chiefly those of a writer and speaker. I was never brought much into contact with him, but he always struck me as a man who was better fitted for Parliament than surgery, and who might have made a considerable mark had his lot been cast where his special talents could have been advantageously displayed.

He died on the 8th of May, 1897, at his country house near Epping, after an attack of influenza.

*James Greig Smith* occupied a leading position at Bristol. He was held in great repute as a surgeon in the city and neighbourhood, and even throughout the west of England, while as a writer his fame was even more extensive. He was held in general esteem, and his untimely death gave rise to a wide and painful sensation.

He was born near Aberdeen, and at Aberdeen educated. He came to Bristol as Assistant House Surgeon to the Royal Infirmary, and rose to be Surgeon to that institution. At first he practised generally, but soon limited himself to surgery, in which he rapidly acquired reputation and practice, and came to be widely resorted to as a consultant in the part of England of which Bristol is a

centre. He was early known as a writer on surgical pathology. He took part in establishing the 'Bristol Medico-Chirurgical Journal,' which for seven years he edited in conjunction with Mr. L. M. Griffiths. In 1893 he was President of the Bristol Medico-Chirurgical Society. He lectured on surgery at University College, Bristol, and his lectures are spoken of as lucid and forcible, and as displaying a mastery of their subject. It is needless to enumerate the lesser writings of Mr. Greig Smith, save that he communicated one paper to our 'Transactions,' but especial mention must be made of his great work on Abdominal Surgery. Of this book, which has taken a leading position in surgical literature, the sixth edition was issued in the present year, shortly after the death of the author. So large a demand for such a book is no small testimony to its merit.

Mr. Greig Smith was not narrowly restricted to surgery, however eminent in this art. He possessed much literary facility ; he was an artist, and was distinguished in several phases of sport.

He died of pneumonia on the 28th of last May, after a very short illness, at the age of forty-three. Short as his life was, it was full of achievement. The work of many years was crowded into a few, and he leaves behind him a reputation such as has been exceeded by few provincial surgeons.

*Dr. Frank Chance* was more distinguished for his knowledge of language than of medicine. He was born in Birmingham in the year 1826. He was educated at Cambridge and at St. Bartholomew's Hospital. At the University, in the year 1854, he became Tyrwhitt Hebrew scholar. Having graduated in due course, he became a Member of the College of Physicians in 1859. In the following year he asserted his connection with medicine by publishing his well-known translation of Virchow's 'Cellular Pathology,' and in 1863 he became a Fellow of the College, the earliest date which the laws permitted. It has always been the custom of the College to have

regard to literary attainment in the selection of Fellows, and on this ground the honour could not have been more worthily bestowed. Had Dr. Chance lived in the sixteenth century it is possible that his fame as a scholar might have ensured his success as a physician; but times have changed, and Dr. Chance found the practice of medicine incompatible with his devotion to the pursuits which he preferred, and in which he eminently excelled. After some experience on the staff of the Blenheim Street Dispensary, Dr. Chance laid aside medicine for philology, took part in translating the Book of Job, and was honoured with a place among the company of revisers of the Authorised Version of the Old Testament. He was little known at our Society, but he gave us the satisfaction of numbering among our Fellows one of exceptional learning.

He died at Nice on the 1st of last July.

*Charles Smart Roy*, Professor of Pathology in the University of Cambridge, was seldom seen within these walls. His chief work, physiological rather than pathological, was brought before the world otherwise than by our means, and the details of his life will be more appropriately considered elsewhere.

Dr. Roy was born and educated in Scotland, and early indoctrinated in physiology and pathology at the Brown Institution, under Dr. Burdon Sanderson. After an interlude as surgeon in the Turkish army during the war with Servia in 1876, he visited the Continent, and spent much time and did much work in the most renowned schools of physiology and pathology. From these he was recalled as the recipient of the George Lewes studentship in physiology, which was awarded to him in his absence. This brought him to Cambridge, to continued physiological work, and led to his selection as Superintendent of the Brown Institute, where he had formerly been a subordinate. In the year 1884, at the age of thirty, he was chosen to occupy the newly established Chair of Pathology at Cambridge, and almost simultaneously was

made a Fellow of the Royal Society. He held his Professorship until his death on October 4th, 1897, at the age of forty-three, after a lingering illness, which for two years had rendered him incapable of performing the duties of his office.

Considering the shortness of his life, the work he did was much. Perhaps the parts of it which are best known are his researches in connection with the heart and circulation, which began at Berlin before the year 1878, and culminated in 1892 with a paper in the 'Philosophical Transactions' in conjunction with Professor Adami. But many other matters, physiological and pathological, occupied his attention, and many gave opportunity for the exercise of his special gifts. Among his pathological inquiries may be mentioned those on the pleuro-pneumonia of cattle, anthrax, the bacteriology of cholera, and the causation of "shock." In relation to physiology, beside his work on the heart were his method of determining the specific gravity of the blood, the seat of the production of hæmoglobin, the formation of lymph, the flight of birds, and the construction of flying-machines.

Professor Roy was an investigator rather than a teacher. He took little interest in the average student whose chief aim was to pass, but he was unsparing in his help to those whose endeavours were directed to research. He possessed great facility in the manipulations necessary to physiological investigation, and had a genius in the invention and application of instruments to this end, of which there are several in use which bear his name. There have been professors who were more attractive to the generality and more widely popular, but perhaps there have been few who could hold him pace in deep experiments. His absolute truthfulness and integrity gave value to all his work, and were apparent in all the relations of his life.

*Dr. Thomas Bell Elcock Fletcher* died on the 21st of last October, at the age of ninety-one. He became a Fellow of this Society in 1842, so that Dr. Fletcher was

one of the seniors of the Medical and Chirurgical. As a Fellow of the College of Physicians he leaves but two who received this honour earlier than himself. He was elected in 1848.

Dr. Fletcher was born at Shifnal, in Shropshire. He received his medical education in Paris, and there graduated. After an interval in London he settled as a physician in Birmingham. In 1838 he was appointed Physician to the General Dispensary, and in 1848 Physician to the General Hospital. He remained on the acting staff of the latter institution for thirty years. He held several other local appointments. He assisted in founding the Sydenham College, a school of medicine which after a time was amalgamated with Queen's College. He at first opposed the amalgamation, but eventually accepted a seat on the Council of the united Colleges.

Dr. Fletcher came to Birmingham without local influence, but he seems to have been a man of great energy, sound sense, and diagnostic acumen, and soon acquired a large consulting practice in and about the town where he resided. I learn that he was humane and charitable, a good townsman as well as a good physician, and that in every way he deserved the high respect which was widely accorded to him.

He retired from practice in 1887, and after a period of repose in the south of France fixed his last abode at Leamington. He had an attack of hemiplegia soon after reaching this place, after which his health was never fully restored. He contributed a paper to the twenty-fifth volume of our 'Transactions' on "Malformation of the Heart."

*Sir Rutherford Alcock*, who died on November 2nd, at the age of eighty-eight, was within a little of being our senior Fellow. He was elected in 1839. There survives one who became a Fellow in the same year, and one who joined in the year preceding, so that two only shared with Sir Rutherford the dangerous honour of seniority.

The career of Sir Rutherford gives an instance of what,

though not unknown, is not very common—the abandonment of the medical profession for another. In my brief notice of his long life I shall not attempt to follow the course of well-known events, or dwell upon circumstances which concern the nation rather than our calling and our Society.

He was the son of a medical practitioner. He was educated at the Westminster Hospital, where he became House Surgeon in the year 1829, at the age of nineteen. He was introduced to the Board by a man well known in his day, but now nearly forgotten—Sir Anthony Carlisle. In 1833 we find him in Portugal as a surgeon in the Marine Brigade, then taking an auxiliary part in the Miguelite and Carlist wars. He rose rapidly, and had attained the rank of Deputy Inspector-General of Hospitals when he retired from the service in 1837, with decorations from the Governments of Spain and Portugal, and medals earned in the field. Now follows an interregnum in which Mr. Alcock, as he then was, acted on a Commission to decide the claims of the British forces with which he had been engaged, and I presume it was at this time that he was appointed Inspector of Anatomy, of which office he was the first occupant. In 1844 he obtained the position of Consul at Fu-chau, and now began the second or diplomatic chapter of his life. He was successively removed to Shanghai and Canton, and in 1858 received the more important post of Consul-General to Japan, which was exchanged in the following year for that of Minister. His achievements in this responsible and perilous office are matters of history. It may be said that the changes which have taken place in recent years in the condition of Japan, the abolition of the dual government, the suppression of military misrule, and the exchange of insecurity and violence for good order and domestic tranquillity are largely due to the judicious and determined conduct of our representative. For his services in this capacity Mr. Alcock received the K.C.B. In 1865 Sir Rutherford, as he now must be styled, was

appointed to Peking, where the character of his administration was expressed by the saying that "the mandarins had found a master."

In 1871 Sir Rutherford retired from the diplomatic service to enjoy not so much rest as change of occupation and independence. He returned to the haunts of his youth, and busied himself in the management of the Westminster Hospital, of which he became a member of the House Committee and afterwards Vice-President, with the frequent position of Chairman of the Weekly Board. I learn from a distinguished member of the medical staff that Sir Rutherford was earnest and active, and worked with a single view to the benefit of the institution ; that he was straightforward, not open to solicitation nor influenced by personal motives. In his relations to the medical staff, while never blindly nor always supporting them, he showed for the most part due regard for any opinion which was widely expressed by the medical members of the Board. Personally he was courteous and warm-hearted.

Apart from his hospital work he was busily and usefully employed in other ways. He was one of the British Commission for the Paris Exhibition of 1878. He was a member of the Royal Commission for inquiring into the London Hospitals for Smallpox and Fever. He was once President of the Geographical Society. He was many times an author, one of the earliest of his productions having been a paper in the twenty-third volume of our own 'Transactions,' on "Injuries of the Joints." Probably his best known work is one which relates his experiences in Japan, and is entitled 'The Capital of the Tycoon.'

In every one of the diverse positions which he occupied he acquired respect and distinction. Addison observes that it is more difficult to fill with credit a life of leisure than one of business, but Sir Rutherford appears to have been equally successful in both endeavours.

In the death of *Dr. James Cagney* we have to lament the  
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too early loss of a physician of some achievement and much promise, who might, had time been afforded, have attained considerable distinction in his chosen line of practice.

He was born in Cork of a Catholic family, many of the members of which had belonged to the Army Medical Service. After a successful career as a student and two voyages as a ship's surgeon he came to London, and worked at St. Mary's and St. Thomas's Hospitals. At the former he devoted himself especially to neurology and electro-therapeutics, and on the retirement of Dr. De Watteville he succeeded him in charge of the Electro-Therapeutical Department. At this hospital he also held the office of Demonstrator of Anatomy, which must have worked in conveniently with his pursuits as a medical electrician. Beside these duties at St. Mary's Hospital Dr. Cagney was connected with two others. He was Physician to the Hospital for Epilepsy and Paralysis, Regent's Park, and Assistant Physician to the North-west London Hospital. But this by no means exhausts the sum of Dr. Cagney's activities. He was at one time secretary to three medical societies. He was a surgeon of volunteers, a golfer, and a sportsman. He was the author of several papers on peripheral neuritis, and I learn that he had made preparations for a small book on this subject. He contributed a paper to the seventy-third volume of our 'Transactions' on the "Mechanism of Suspension in the Treatment of Locomotor Ataxy."

Enough has been said to show that Dr. Cagney's life, though short, was productive, and was crowded with diverse interests. He appears to have made many friends, and to have been respected as well as liked.

He died on November 18th of typhoid fever, in his thirty-eighth year.

*Henry William Freeman* died on the 21st of last November, at the age of fifty-five. He was well known at Bath, where he had acquired a large practice and much local prominence.

Mr. Freeman had originally no connection with Bath ; he was born and received his early education in North Devon, his medical education at the Middlesex Hospital. His association with Bath began soon after he became qualified, when, at the age of twenty-two, he obtained the post of Resident Medical Officer to the Royal United Hospital. After a year he resigned this office and set up in private practice, in which he early met with much employment. In 1881 he was elected Honorary Surgeon to the hospital where he had formerly been Resident, and at once began to devote himself to operative surgery, in which, as I learn, he was very successful.

He now turned his attention to the improvement of the baths, and visited many Continental and American watering-places in search of information to this end. He was made mayor, and in that capacity took a prominent part in the ceremonious opening of new baths in 1889. He seems to have been greatly interested in the city of his adoption, and would have presented a house for a convalescent home had means been forthcoming for its maintenance.

It was observed by one of the wisest of our contemporaries that one engaged in the exacting pursuit of medicine should not allow himself to be wholly engrossed by it, but should encourage some special interest outside his own profession ; in other words, he should have a hobby. We have within our ranks, or have recently had, men distinguished as artists, astronomers, geologists, collectors of rare books, and sportsmen of various kinds. Mr. Freeman was eminent as a breeder of race-horses. Among his other pursuits he found time for authorship, and wrote a book on the Thermal Waters.

He died after a short illness comprising cardiac failure and anæmia. He was at work up to within ten days of his death.

I now have to notice the demise and the career of a great provincial surgeon.

*Edward Lund* was born at Peckham in the year 1823,

apprenticed at Faversham, and afterwards educated at Guy's. After becoming qualified he went to Manchester, and soon became connected with the Pine Street School of Medicine, first as Demonstrator, then as Lecturer on Anatomy. On the amalgamation of this school with that in Chatham Street Mr. Lund became a member of the teaching staff of the Manchester Royal School of Medicine. In 1868 he became full Surgeon to the Royal Infirmary, where he had already held a junior post; and ultimately, on the union of the Medical School and Owens College, he became first joint Professor, then sole Professor of Surgery. His teaching in this important position is said to have done much to establish, or perhaps I should say to maintain, the reputation of the Manchester school. Mr. Lund early recognised the use of antiseptics in surgery, and preached the Listerian gospel *in partibus infidelium*.

Not only at Manchester was Mr. Lund honoured and influential, but, to pass over lesser distinctions, he was chosen in 1881 as a member of Council of the Royal College of Surgeons, and was afterwards made Examiner and Hunterian Professor. These marks of esteem, great in any case, were especially significant when conferred upon a surgeon whose centre of activity was in Lancashire, and it is a testimony to his energy that his duties at the College were, as I am told, punctually and conscientiously performed.

Mr. Lund did nothing for our Society, but was the author of several papers in the journals on surgical subjects.

He died on the 4th of last February of cardiac failure and pulmonary congestion.

The course of his life and the honorable position to which he attained are sufficient evidence of the respect in which he was held, not only in his own locality, but generally by the profession.

Of *Dr. Leonard Remfry* I can speak from personal intimacy, which dated from his entry as a student at

St. George's Hospital in 1882. Descended from an old Cornish family, he was born in Calcutta, the son of an East India merchant. His general education was obtained at Dulwich and Christ's College, Cambridge. At St. George's he obtained in succession, not to mention minor appointments, the offices of House Physician and Obstetric Assistant. He was universally respected and liked. His good-nature, kindness, and geniality made him many friends, and, indeed, it may be said that these included all with whom he came into habitual contact. His work was always conscientiously and well done, and the esteem in which he was held was shown in 1894 by his unanimous election to the newly created post of Assistant Obstetric Physician. He had already been appointed Obstetric Physician to the Great Northern Hospital, and his line of practice fixed. His qualities could not fail to command success, and, indeed, practice was fast gathering around him when his hopeful career was cut short by his untimely death.

Dr. Remfry was the author of several papers, of which the subjects of the more important may be briefly indicated : pulmonary embolism, saline injections into the veins in collapse after ovariectomy, the effects of lactation on menstruation and impregnation, division of the broad ligaments and ovariectomy over eighty.

One who was especially connected with Dr. Remfry in hospital work speaks of his excellence as a teacher, and of the respect which was inspired by his entire single-mindedness and straightforwardness, qualities with which he will readily be credited by all who knew him.

His sudden death at the age of thirty-seven, as the result of an epileptic attack, came as a shock upon his colleagues and numerous friends, by whom his memory will long be held in affectionate regard.

Of *Dr. William John Vereker Bindon* I have been able to learn only that he joined our Society as a Non-resident Fellow in 1878, that he was formerly in general practice in Manchester, and that he recently died in Australia.



A CASE  
OF  
CALCAREOUS METAMORPHOSIS OF THE  
TRACHEAL LYMPHATIC GLANDS

FORMATION OF MEDIASTINAL ABSCESS AND ESTABLISHMENT  
OF BRONCHIAL FISTULA; SUBSEQUENT PERFORATION  
OF THE WALL OF THE ARCH OF THE AORTA; LONG-  
CONTINUED REPEATED HÆMOPTYSIS;  
DEATH FROM HEMIPLEGIA

BY  
SIDNEY PHILLIPS, M.D.

PHYSICIAN TO ST. MARY'S HOSPITAL; SENIOR PHYSICIAN TO THE  
LONDON FEVER HOSPITAL

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P. M—, aged 26 years, had always been delicate. His father and father's family were excessively gouty; his mother suffered from rheumatism. He had had no illnesses of any note, but at about the age of eighteen he suffered from frequent profuse epistaxis, for which he went on a sea voyage. He was very subject to attacks of tonsillitis. During the year 1885 he experienced a good deal of pain in the elbows, wrists, and finger-joints; the larger of these

joints contained little fluid, but on movement very rough grating could be felt within them. He went as a cadet to the Royal Military College at Sandhurst in 1886, and during the summer vacation of 1887 he went to Nuremberg; he complained much of the foul smells there, and was rendered quite ill by them: this was in August. He returned to Sandhurst fairly well at the end of that month, and while riding in the school there on September the 5th, 1887, he was suddenly attacked with very acute pain on the right side of the chest; this became more and more severe, and he consulted me in London on September the 11th. There was then intense pain round the right side of the thorax, almost limited to the third intercostal space, and with it extreme hyperæsthesia of the surface of this side of the chest as far down as the sixth rib; no tenderness existed over the spine; the temperature was normal; his general condition seemed good, and I could find no evidences of disease of any part of the lungs or the heart. I ordered him salicylates internally, and locally anodyne applications.

He returned to Sandhurst, but the pain grew more and more intense, confining him to bed, and morphia injections had to be administered by Brigade-Surgeon Clark, who attended him then and during the subsequent illness.

On September the 16th—eleven days after the pain had commenced—he had a severe rigor, and the temperature rose to  $102^{\circ}$ .

On the next day I saw him with Dr. Clark; his tongue was coated, his liver a little enlarged; the pulse was full and bounding, the lungs appeared to be absolutely healthy; there was a suspicion of a soft systolic murmur over the aortic cartilage. Beyond this there were no indications of disease anywhere, but his temperature was  $104.6^{\circ}$ , and the pain was very severe.

During the next five days he improved a little, the temperature gradually falling to normal, and the pain lessening, but on September the 22nd—six days after the first rigor—rigors recurred, and again on each of the six

succeeding days, each time with a rise of temperature to  $104^{\circ}$  or more. Between the rigors the temperature fell to  $99^{\circ}$ .

On the 27th September I saw him again: he had just had a rigor with a temperature of  $105^{\circ}$ , accompanied by agonising pain in the fifth, sixth, and seventh left intercostal spaces just outside the cardiac area. The pain was intense, and he screamed with agony. There was a great reluctance to inspire, and he kept the painful side of the chest almost motionless, only a faint wheeze being audible as a breath-sound. There was now tenderness over the spines of the fourth and fifth dorsal vertebræ. There was a faint friction crackle just below the lower angle of the left scapula. The pulse was 145 a minute, very feeble. He had taken food well, but had lost flesh greatly. Morphia had to be repeatedly administered, and he took quinine at times.

On the 29th September he was seen by Sir William Broadbent, with Dr. Clark and myself; he then looked better, the pulse was 84 a minute, and the temperature  $99^{\circ}$ . No sign of disease anywhere could be detected, except the faint friction sound over a spot in the left back, and air did not enter so well on this side as on the right, though on deep inspiration it entered more freely, suggesting some pressure upon the left bronchus. There was no recurrence of rigors after the 27th September, but the profuse sweats and daily rises of temperature continued, with pain and loss of flesh and strength. The friction sound extended in area, and by the 8th October it was audible over the left back as high as the spine of the scapula, and round the axilla; cough was slight; heart-sounds normal.

About the 15th October there became superadded to his troubles a most incessant, violent, and exhausting spasmodic dry cough, preventing rest or sleep. Laryngoscopic examination showed the larynx to be normal. The pyrexia now again increased, reaching  $103.4^{\circ}$  on October the 19th, when he was seen with us for a second time by



Sir W. Broadbent. The friction sound was now audible all over the left back, and the cough kept him constantly sitting up in bed, and he was exhausted to an extreme degree.

On October 28th the cough became more violent than ever; it was apparently due to some irritation of the laryngeal nerves. On this day there suddenly came on signs of rapid copious effusion into the left pleura. The apex of the heart became at once pushed to the right, and could be felt well outside the right nipple line; its action was greatly disturbed. Simultaneously there developed loud systolic murmurs over the base of the heart. I saw him that afternoon, and the orthopnoea being great we at once aspirated the left chest, withdrawing 56 ounces of clear fluid. No attempt was made to withdraw all the fluid in the chest. The aspiration gave considerable relief, respirations falling from 38 to 24 per minute, and the pulse improving.

By October 30th the fluid had re-accumulated, the respirations were 40, and the pulse 140 a minute. His distress being great, 20 ounces more fluid were removed by aspiration; more was not removed as the patient was under chloroform, and his condition was very critical; after this the respirations fell to 22 a minute, and he was much easier.

On the next day the temperature was only  $101.2^{\circ}$ , the respirations 32, and the pulse 140 a minute. During the night of November 1st he had an attack of syncope with complete unconsciousness and flickering pulse; this lasted three hours. He was thought to be dying, and his relatives were summoned; but he was treated by Dr. Clark with subcutaneous injections of ether, and gradually recovered consciousness.

On the next morning 10 ounces more fluid were removed by Dr. Clark with the aspirator. The temperature was  $102^{\circ}$ , and the cough incessant.

The same afternoon, November 2nd, he expectorated after violent cough about one drachm of pus slightly

tinged with blood and mixed with aërated mucus; this was the first expectoration of any sort throughout the illness.

On the next day the cough had become a little less, but there was still great pain over the third and fourth left intercostal spaces.

I saw him on that afternoon, and there was a great change in the physical signs on the left side of the chest; there were still all the signs of pleuritic effusion over the back, but in front of the chest there was now audible loud tubular breathing. This extended round to the axilla, and there were sibilant râles of a resounding character, as if in some large cavity; there was dulness on percussion over the area of tubular breathing; it appeared probable there was some air in the pleural cavity, but the physical signs were anomalous. The heart's apex was now just below the right nipple.

Although the cough had somewhat decreased in intensity the patient continued to lose ground, with daily rises of temperature to  $102^{\circ}$ , and considerable dyspnœa and pain.

On November 6th, the dyspnœa having rather increased, Dr. Clark removed eighteen more ounces of clear fluid by the aspirator.

On November 9th the temperature was a little lower, and the pain and cough were less; there seemed to be less serous effusion in the pleura, but there was a peculiar "crack-pot" sound on percussion just below the inferior angle of the scapula; the tubular breathing in front of the scapula had now given place to loud amphoric breathing, but no gong sound or succussion sounds were obtainable: the heart's apex was still to the right of the nipple line, and the murmurs, which had for a time disappeared, returned—a loud systolic murmur behind the sternum at the level of its junction with the second and third rib cartilages, and in the second and third intercostal spaces; there was also a very loud bruit at the angle of the left scapula: the patient's breathing was again greatly embarrassed, and his pulse very rapid and compressible.

On the next day, the 10th November, he had another syncopal attack of ten minutes' duration.

On November the 11th the temperature was  $103.6^{\circ}$ , and aspiration in the sixth space failed to remove any fluid; on the same night he had another very severe syncopal attack.

The patient had now for a period of two months suffered from terrible pain, from protracted fever, and latterly from a most exhausting cough; his rest had been greatly interfered with; he was emaciated to a skeleton, and becoming weaker daily; he had been aspirated eleven times, and though the serous effusion in his chest did not re-accumulate to any extent, the heart remained displaced, the fever continued, and syncopal attacks were becoming more frequent,—he had had two in the last thirty-six hours; he was at times delirious, and it appeared certain that he might die at any moment from syncope, or would gradually sink from exhaustion unless relieved by treatment.

Mr. Godlee was therefore asked to see him, with the view of making a free incision into the chest wall.

On November the 12th A.C.E. mixture was administered by Surgeon-Captain Mansell, who had aided us in the care of the patient throughout the illness, and Mr. Godlee made an incision into the left axilla in the fifth intercostal space. About ten ounces of clear fluid ran out; the finger passed into the wound entered a very large cavity, the diaphragm being felt below, and the lung being greatly collapsed and retracted, with sticky lymph upon its surface. A very large drainage-tube was inserted and the wound dressed with salicylic wool.

After this improvement commenced, the temperature falling to  $99.5^{\circ}$  by the 30th November; the cavity, explored with a bougie, appeared to be of enormous size, and discharged very freely; at times drainage was difficult, and on October the 2nd the temperature rose to  $102.2^{\circ}$ , the pulse-rate to 140 a minute, and the patient became almost delirious. The incision was enlarged a

little, and a good flow of pus resulted. The cough and pain were now but slight, but there was still some tenderness over the fifth and sixth dorsal spinous processes. By the end of December he was able to be got up, and he travelled to Bournemouth in an invalid carriage on January the 3rd, 1888, under the superintendence of Dr. Clark, to whose unremitting attentions his recovery had been mainly due.

At Bournemouth he was under the care of Dr. E. Hyla Greves, with whom I saw him on several occasions. The cavity continued to gradually decrease in size, but attempts to discontinue the drainage-tube were always followed by difficulties, and it had to be replaced. He became strong enough to walk a little, and came to London on March 20th, 1888; improvement continued, and he went to the country again at the end of May for a short stay, but quickly returned with pyrexia and retention of pus in the cavity; good drainage was soon re-established. About this time, while exploring the cavity with a long bougie, I once felt at the tip of it something feeling rough, like a small surface of carious bone. It appeared far back near the spinal column, but I was never able to hit upon it again, and it was not until the necropsy that this rough surface was shown to be one of the calcareous mediastinal glands.

Mr. Godlee saw the patient with me once or twice, and as the opening could not be closed, an anæsthetic was administered, and Mr. Godlee removed two or three inches of the fifth and sixth ribs, which had become united together by two cross-pieces of bone, forming a complete osseous ring around the drainage-tube: after this the chest wall fell in well, and by the middle of June the drainage-tube had been discontinued, and the opening in the chest completely closed—six and a half months after the incision into pleural cavity.

This terminates what I may call the first chapter of the patient's illness.

Then followed a period of eighteen months of quiescence

of symptoms, though never of perfect health. The patient gained flesh and strength, and was able to enjoy himself, but he always suffered from what he termed "asthma," a wheeziness which became very marked in damp or foggy weather; the voice, too, at times was a little husky, but it had not lost its power, and he was able to sing; there was no cough, dyspnoea, expectoration, or fever, and the only physical signs of disease within the chest during the beginning of this period were those due to the old pleurisy.

In November, 1888, he went to Australia, and came back early in 1889 much stronger, but there was still a little wheeziness and huskiness of voice. Shortly after he came home—in June, 1889, he brought up one day a little semi-purulent pink-stained matter, much resembling crushed strawberries and cream; there were no tubercle bacilli found in it. Below the right clavicle I noted on the next day that there was a slight rise of percussion pitch and a little weakening of air entry.

In November, 1889, he went to Cairo for the winter; he continued very well there till 18th January of 1890. On that day commenced the third epoch of the disease.

He had on the 18th January, 1890, got wet while out on a shooting expedition, and on the next day he suddenly, without effort or cough, brought up about seven ounces of bright red blood. He then came under the care of Dr. H. Sandwith of Cairo, who kindly communicated to me from time to time the progress of the case. The hæmoptysis recurred again and again at intervals of a few days; the longest interval was three weeks, and then it came on immediately after he had been carried to the roof of the hotel for fresh air; any exertion or a fit of laughter would bring on the hæmorrhage. Dr. Sandwith found over the apex of the right lung some wasting of the pectoral muscles, slight deficient percussion resonance, and increased vocal resonance, with an occasional moist sound.

In the middle of April, being somewhat better, he was brought homewards by sea to Marseilles, and thence by

rail to Cannes ; on the sea journey he had several attacks of hæmorrhage, and had to stop at Cannes, where he was seen by Dr. Battersby and Dr. Bright.

He arrived in London on May 21st, and came again under my care.

I found the same slight physical signs as Dr. Sandwith had noted over the apex of the right lung, the most marked abnormality being the increase of vocal resonance over the supra-spinous fossa. Tenderness still existed over the sixth dorsal spine.

The attacks of hæmorrhage continued ; the blood was always bright red, unmixed with air, sometimes partly clotted ; it sometimes came up in a sudden gulp when he was walking in the street, at other times it woke him up from sleep ; after it had been gulped up there was an end of it for the time, and no trace of blood came up till the next free hæmoptysis. The usual quantity of blood ejected was seven or eight ounces, sometimes as much as ten ounces, and at one time the bleeding was much greater over two successive days. The blood was repeatedly examined, but no tubercle bacilli could be found in it by Mr. Watson Cheyne or others. The hæmorrhage recurred at irregular intervals, on an average twice a week. It did not seem to be determined by exertion, and though any very active exercise was forbidden, it was thought best to let the patient take daily walking exercise.

At times soreness about the larynx was complained of, but Sir Felix Semon, who examined him, found the larynx normal. There was no expectoration, except that in the intervals of the hæmorrhages he coughed up frequently tube-like casts of the smaller bronchial tubes ; they were always quite white and unstained by blood ; they often excited considerable cough on ejection. Microscopically I found these casts to be tubular, non-laminated, and of homogeneous, apparently fibrinous composition.

In May, 1890, while at Eastbourne under the care of Dr. Downes, he once brought up some small pieces of whitish pellicle, looking very like hydatid membrane, but

the microscope did not show any lamellated structure, and this never recurred.

The physical signs remained the same as I have described, but there were slight differences on different occasions when I examined him, the dulness sometimes quite disappearing, and occasionally distant weak tubular breathing was audible. During this period of the illness he was seen once again by Sir William Broadbent ; also by Sir Richard Quain, and by Dr. James Pollock and by Mr. Godlee.

For fifteen months the hæmorrhages continued ; he certainly lost on an average 16 ounces of blood a week, and in all the quantity lost must have been nine or ten gallons of blood. He recuperated after each hæmorrhage with extraordinary rapidity, and showed great spirit and courage in making the best of his troubles ; but after about a year it became obvious that he was failing to make good the repeated drain of blood ; he became anæmic and sleepless, with a hectic flush and transparency of skin, and the pulse was very soft and compressible : there was never any fever, and he always ate well.

In the winter of 1890 he went to Cannes again, but soon returned, the hæmorrhage having become more profuse.

On the 26th of March, after two days of very free bleeding, he became hemiplegic on the left side ; unconsciousness supervened, and he died in this condition on April 2nd, 1891.

The *post-mortem examination* made by Dr. L. Guthrie and myself showed that all the organs were healthy. Nothing abnormal was found in the heart or pericardium ; there was little to denote the previous existence of the opening in the left chest except fibrous adhesions of the lung. The apex of the right lung appeared slightly shrunken, and felt a little tougher than the apex of the left ; there were no tubercles anywhere in the body. In the tracheal bifurcation was an ovoid mass the size of a walnut, adherent by connective tissue to the bronchi and the trachea, and con-

sisting of a group of lymphatic glands which had become calcareous and brittle, but which were still distinct in outline. They were united together by tough areolar tissue, leaving a small irregular cavity in the centre of the mass they formed. On slitting up the air-tubes the larynx was healthy, and a small orifice was seen in the inner aspect of the right bronchus, half an inch from its commencement. A fine probe introduced through the orifice passed into the cavity in the centre of the calcareous mass; the under surface of the commencement of the transverse part of the arch of the aorta touched the upper part of the rough calcareous glands, and at this spot there was a small perforation through the wall of the aorta into its lumen; a probe could be passed through this orifice in the aorta into the centre of the gland mass, just as it could be from the opening in the bronchus; the cavity in the calcareous mass was thus in communication on the one side with the interior of the aortic arch, on the other side with the interior of the right bronchus; it was in this way that the hæmoptysis had occurred. The ascending aorta was extremely small in circumference, resembling that of a child,—no doubt from the diminished volume of blood in the body which resulted from the long-continued losses of blood. Two or three isolated bronchial glands had undergone cretaceous changes.

*Remarks.*—Briefly, the course of events in this case appears to have been—

I. Calcareous metamorphosis and matting together of a group of lymphatic glands, forming a rigid tumour attached to the tracheal bifurcation.

II. Acute inflammation and suppuration in the connective tissue of this tumour, forming an encysted abscess.

III. Perforation of the right bronchus by pus from this abscess, leaving a permanent bronchial fistula.

IV. Subsequent erosion and perforation of the wall of the arch of the aorta by pressure of the calcareous glands.



V. Recurrent hæmoptysis from the aorta into the empty abscess cavity, and thence through the bronchial fistula into the trachea.

VI. Death from hemiplegia, the result probably of thrombosis from the effect of the repeated hæmorrhages.

Whether or not this was the actual sequence of events, the calcareous changes in the glands, the suppuration and formation of a bronchial fistula, the perforation of the aorta, and the repeated hæmorrhages from it are beyond question.

It is to these points I wish to draw attention, but it would be impossible within the limits of this paper to make any attempt to review the literature bearing on the many interesting clinical and pathological conditions which presented themselves during the three and a half years' duration of the case.

#### *Calcification of the Mediastinal Lymphatic Glands.*

Calcareous bronchial glands are occasionally found at post-mortem examinations of old persons, or in those in whom exist old tubercular foci in the lungs. In the present instance, however, a whole group of glands underwent this calcareous metamorphosis, and became united together by the periglandular areolar tissue into a hard unyielding mass situate in the bifurcation of the trachea, and adherent to it by fibrous tissue. Medical literature affords but scanty information as to the causes which lead to calcareous changes in the lymphatic glands, but they are probably the same as lead to similar changes in the walls of arteries and of other tissues. The patient whose case I record this evening was young, and was not tubercular, but he had shown a great tendency to arthritis with rough grating in the joints; and it seems probable that the calcareous changes were due to the rheumatic or gouty diathesis, the deposit being determined in the lymphatic glands of the chest, owing to some idiosyncrasy on the part of the patient. The changes in the glands and

the formation of the tumour must have been very gradual, and no symptoms arose until acute inflammation chanced to be set up.

I have not found any record of a similar condition ; the only case in any way similar to it that I know of is one alluded to by Dr. Henry, in the 'Transactions' of this Society for the year 1819, in which the autopsy of a girl of fifteen, who had suffered from much cough, revealed a concretion the size of a musket ball embedded in the left bronchus. But though this case has a superficial resemblance to the present one, it appears to have been really of quite a different nature ; for, as far as can be judged from the report of Dr. Henry's case, the mass was not of glandular origin, and was formed within the bronchial tube, and did not invade it from without.

#### *Formation of Mediastinal Abscess.*

Mediastinal abscesses usually have their origin in disease of the vertebræ or in scrofulous lymphatic glands. In the present case there was no such cause, and it seems probable that while the patient was riding a sudden movement or strain lacerated or tore some of the areolar tissue which matted the glands together, or even fractured one of the brittle glands, setting up an acute inflammatory process which went on to suppuration. Suppuration in the mediastina is usually diffuse, but one remarkable point in the present case is that the formation of pus took place within a pathological cavity with rigid walls which encysted it, until it eventually made an exit for itself by perforating the wall of the right bronchus to which the tumour was adherent.

The symptoms which occurred during the patient's illness up to the date when this occurred may be conveniently considered here.

The first symptom was the *pain* around the wall of the chest ; it was at first felt on the right side, afterwards also on the left. It was accompanied by extreme hyper-

æsthesia of the surface of the chest wall, and by tenderness over the fifth and sixth dorsal spinous processes. The pain itself was most intense, and was insupportable without repeated injections of morphia; it lasted for many weeks, and until the tension of the abscess was relieved by the discharge of pus into the interior of the bronchus. There was considerable tenderness over the vertebral spines, but the pain could not be attributed to disease of the vertebræ, for there was free movement of the spinal column, and no rigidity of the muscles of the back.

So long as the pain was the *only* symptom its cause was quite unascertainable, but I think that the post-mortem appearances prove that it was a pain referred to the chest wall from the seat of disease in the mediastinum, and was set up there by the tension of the pus imprisoned within the rigid walls of the calcareous tumour. Pain around the chest wall is similarly complained of in some cases of aneurism of the arch of the aorta when it is not possible that it can result from any actual pressure upon the intercostal nerves. The tenderness over the vertebral spines was at the same horizontal level as the mass of calcareous glands, and was probably dependent upon it. This transference of pain to the intercostal spaces and the tenderness over the spinous processes from a lesion in the centre of the chest cavity may possibly be of service in the diagnosis of future obscure cases of mediastinal disease.

After the pain had persisted for days there occurred repeated rigors with intermittent severe pyrexia, and it became evident that there was organic disease somewhere within the thoracic cavity. Of lung disease there was still no sign; the absence of murmurs (except the slight systolic murmur heard at the base of the heart on the first day), the intensity of the pain, and the tranquillity of the heart's action militated against ulcerative endocarditis or endarteritis, and we came to the conclusion that the fever and rigors were only to be attributed to pus formation, and that the pus was probably in the mediastinum; its exact locality remained a mystery

until the supervention of the incessant and harassing spasmodic cough, which, in the absence of any laryngoscopic sign of disease, pointed to something close to the air-passages, and causing irritation of the laryngeal nerves. It was still, however, inexplicable that we could find no other pressure signs or other indications of the exact locality of the pus. The necropsy showed that the abscess was encysted within the calcareous tumour. It was this imprisonment of the pus in a previously formed pathological space, the existence of which had given no symptoms, that made the exact diagnosis so impossible. No doubt the incessant cough was set up by the gradual perforation of the bronchial wall by the abscess.

The *attack of pleurisy* commenced apparently over the lower part of the back of the left lung, for it was here that localised pleuritic friction sound was audible for some days. At the time it was difficult to explain why the pleurisy occurred, and why it started at a distance from the local disease in the mediastinum; and even with the knowledge subsequently acquired from the post-mortem examination the reasons for the pleuritic attack remain unexplained.

After the dry pleurisy had lasted for some days there suddenly succeeded a very rapid and copious serous effusion; the heart became at once greatly displaced, and loud murmurs became simultaneously audible; the patient's life was in imminent danger, and death was certainly averted by the first aspiration; after repeated aspirations the effusion gradually became less and less.

*Pneumothorax*.—After the serous effusion had gradually lessened till its quantity was but small, the heart still remained displaced, and the syncopal attacks became more frequent. At about this time there were indications of free air in the thoracic cavity, but the amphoric breathing which denoted this was limited to the front of the left chest, while there was serous effusion at the back on the same side, as shown by the physical signs and by

the aspirator. Again, though there was air and there was liquid in the left side of the thorax, no succussion sound could ever be obtained. The physical signs, therefore, were somewhat anomalous, and no air passed out through the aspirator when it was introduced into the region of the serous effusion.

At the time I thought that air must have entered the chest during the aspirations. With the light thrown upon the case by the necropsy, it seems more probable that it was sucked in with each inspiratory act through the fistulous opening in the right bronchus, and accumulated until it came to exercise considerable pressure upon the pericardium and produced the later syncopal attacks.

Again, I am at a loss to account for the presence of air on the *left* side of the chest, while the opening was in the *right* bronchus; but it must be remembered that the relative position of organs was greatly altered by the extreme displacement of the heart to the right by the serous effusion before the perforation in the bronchus had occurred.

Is it possible that the air which entered through the opening in the bronchus did not pass into the pleural sac, but into the mediastinal space, and forced its way between the left edge of the pericardial sac and the left pleura which had become adherent? This might account for the anomalous physical signs of pneumothorax to which I have above directed attention. Be this as it may, I think the repeated syncopal attacks and the physical signs I have described are of interest as resulting from pressure of air within the chest cavity, after the quantity of serous pleural effusion had become insignificant.

During the second and third periods of the patient's illness there existed a *fistulous opening through the right bronchus*. The only symptoms it gave rise to were a little wheeziness on breathing and a slight huskiness of voice, both much increased by damp or foggy weather; the small tree-like casts of the bronchial tubes were not expectorated until after the communication with the interior of

the aorta had been established, and they cannot be attributed solely to the presence of the bronchial perforation. It was not till several months after this perforation had been produced that there was any deviation from the normal detectable in the physical signs, but eventually there was a slight flattening of the upper part of the right chest and a rise of pitch in the percussion note, with a marked increase in the vocal resonance.

When the hæmoptysis became associated with these signs the case greatly simulated one of phthisis; the post-mortem examination, however, showed no trace of phthisis, and proved that the physical signs were produced by the slight shrinking of the apex of the lungs due probably to its not being for a long period fully distended on inspiration, in consequence of the perforation in the bronchus. The clinical simulation of phthisis by the bronchial fistula is one of the interesting features in this case.

*The hæmorrhage from the aorta.*—From the preparation (1148, St. Mary's Hospital Museum) which I show this evening it will be seen that the perforation of the wall of the aorta is situate in the beginning of the transverse part of the arch of the aorta.

No doubt it resulted from gradual erosion of the wall of the artery by its contact with the calcareous glands, a result which must have been materially assisted by the respiratory movements of the trachea to which the calcareous mass was attached, and by the repeated distension of the aorta itself with each systole of the heart. The opening into the aorta led into the interior of the calcareous tumour, whence the blood passed into the air-passages through the perforated bronchus. The amount of blood lost by the patient during the fifteen months of recurring hæmorrhages from the aorta must have amounted to not less than nine or ten gallons; but more extraordinary perhaps than the amount of blood lost was that the patient continued to live with a free

opening into the aorta distant only three inches from the heart itself.

The *diagnosis* at this period was replete with difficulties. As already pointed out, there were certain physical signs at the apex of the right lung which, taken with the hæmoptysis, suggested tubercular phthisis, but as time went on these physical signs in no way increased, no tubercle bacilli were ever discoverable in the blood ejected, and the amount of blood lost was far more than was consistent with the slight phthisis that was present if there was any phthisis at all. In addition there was no fever, and the patient picked up strength immediately after each hæmorrhage with a rapidity which made it certain that it was not merely a case of phthisis to deal with.

At one time some small pieces of membrane were expelled, and this with the repeated hæmorrhages and slight physical signs gave the case a resemblance to one of hydatids of the lung, but no more membrane was afterwards brought up that might have been microscopically examined.

Aneurysm was excluded by the absence of bruit or of pressure signs, except that I on one occasion heard over the right supra-spinous fossa a faint distant sound like a venous hum. The exact condition remained a mystery until the autopsy revealed the remarkable coincidence of openings into the aorta and bronchus which it would have been quite impossible to foretell.

The repeated expectoration of the *bronchial casts* is the only other point on which I need comment; they were always preceded by hæmoptysis, usually by a few hours only, but they were never in the least blood-stained. They showed no laminated structure under the microscope, such as is seen in some cases of plastic bronchitis, and the only supposition I can form as to their origin is that they resulted from coagulation of the fibrin of some of the blood which escaped into the smaller bronchial tubes.

I know of no case at all resembling the present case except one in the 'Transactions' of this Society for 1846, reported by Mr. Busk.

A woman aged 35 had a small hard lump in the mesial line of the neck, close above the upper edge of the sternum. This gave no pain or inconvenience for thirteen and a half years; the swelling then suppurated and opened spontaneously, discharging a quantity of "white matter." Five months later bleeding came on suddenly from the fistulous opening; this was arrested by a compress, but recurred the next morning. On removal of the compress the blood was ejected in a jet which rose above the surgeon's head. The bleeding continued, and the woman died in forty-eight hours from the commencement of the hæmorrhage.

The autopsy showed that the ulcerated opening communicated with an old abscess cavity containing a pound of coagulum occupying the point of the neck below the thyroid cartilage, being bounded posteriorly by the trachea, in front by the fascia and integuments. The cavity extended downwards and backwards between the right bronchus and the arteria innominata, behind the root of the right lung to the front and right sides of the bodies of two or three upper dorsal vertebræ, and inferiorly the abscess cavity reached the right side of the ascending part of the arch of the aorta, and for about two inches below the origin of the innominate artery, the external cellular tunic of that vessel as well as the greater part of the external side of the arteria innominata was completely removed. The middle coat of the aorta was quite bare, and there was a small opening or fissure about one eighth of an inch long in the middle of the denuded portion opening into the main trunk of the vessel.

This case resembles that I have recorded this evening in affording, to use Mr. Busk's words, "an unequivocal instance of a communication between the cavity of an abscess and the aorta, in consequence of an ulcerative process set up from without."



But there are many points of difference between the two cases ; and what makes the present case I believe a unique one is the formation of the calcareous tumour which subsequently became the medium of communication between an opening into the bronchus and an opening into the main trunk of the aorta, so that there eventually resulted the unique condition of an old abscess cavity communicating on the one side with the aorta, on the other side with the bronchus, the blood being thus afforded a free exit into the trachea.

Mr. Busk remarks on the length of time—forty-eight hours—his patient survived after hæmorrhage had commenced from the ascending aorta, but in this particular the present case is far more extraordinary, for a similar hæmorrhage here continued for no less than fifteen months.

With regard to the treatment, it was directed to the relief of the intense pain by the use of morphia and other anodynes. When it became evident that there was pus to be evacuated we were on the watch for any local indications of its presence, and it was eventually evacuated spontaneously. Withdrawal of liquid from the pleura was performed when there was great orthopnœa or tendency to syncope. The terrible cough was very difficult to alleviate, but an oro-nasal inhaler with eucalyptus gave most relief.

The free opening was made into the chest cavity, though there was no sign of the liquid in it being purulent, only after very careful consideration and with the certain conviction on the part of those who had watched the case from the commencement that the patient would very soon die unless it were done. Its results were most satisfactory, for it resulted in the cure of the disease so far as it had then gone.

During the period of recurrent hæmorrhages the only treatment possible was directed towards controlling it by rest and by drugs, of which far the most efficacious proved to be the tincture of hamamelis.

Beyond this, in the obscurity which rested upon the nature of the case, nothing could be attempted, and it is certain that even had we known of the exact conditions present no further treatment would have been feasible.



A FURTHER CONTRIBUTION  
TOWARDS THE  
PATHOLOGY  
OF THE  
PERNICIOUS MALARIAL FEVERS OF  
SIERRA LEONE  
AND  
OUR KNOWLEDGE OF THE PARASITE WITH WHICH  
THEY ARE ASSOCIATED  
BY  
GEORGE THIN, M.D.

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THERE is in the seventy-ninth volume of the 'Medico-Chirurgical Transactions' a note by myself on the appearances found in the tissues in a fatal case of pernicious malaria at Sierra Leone.

In this case the patient died comatose, and I found in the blood-vessels of the brain many parasites of malaria. Although a very few parasites could be found in the red blood-corpuscles in other organs, it was only in the brain that they were found in any number, and there they were

very numerous. Much pigment was found in the liver and spleen, and its disposition in these organs is described in the paper. The epithelium of the convoluted tubes of the kidneys was found much broken down. I pointed out that the appearances which I described coincided with those described by Bignami as being found in fatal cases of malignant malaria in the south of Italy. Since that paper was written I have examined in much detail tissues of four additional fatal cases which occurred at Sierra Leone,—three of them fatal from malaria, and one of them, in order to afford a basis of comparison, from a fatal case of pericarditis. For these tissues I have to thank Surgeon-Captain Duggan, who preserved and sent them to me from Sierra Leone, and both myself and (I may perhaps venture to say) the Society are much indebted to this gentleman for the interest and trouble he took in preparing and sending the specimens.

The subject is, I consider, sufficiently important to justify me in placing before the Society the details connected with a histological examination of the tissues in these additional cases.

In numbering the cases I take them in the order in which they were examined. The case previously described is taken as Case 1. The case of pericarditis, although not described in detail, is mentioned as Case 3.

CASE 2.—E. M— was admitted to hospital on the morning of the 1st of March, 1896, complaining of headache and pains all over the body. He had the appearance of a man who had been drinking heavily. His tongue was brown and dry and tremulous, and his hands were shaky. He denied that he had been drinking. His temperature was  $103^{\circ}$ , and his pulse good. Five grains of antifebrin, five grains of calomel, and twenty grains of quinine hydrochlorate were given. In the evening his temperature was  $102^{\circ}$ , and his pulse good. He had sweated freely, but his skin was again dry.

His bowels had been freely opened. The antifebrin was ordered to be repeated. During the night he was restless, and complained that he could not sleep. At 5 a.m. he got up and went to stool. At 6.30 a.m. he was comatose. Temp. 100°. On the arrival of Surgeon-Captain Thomson he had expired.

One of the orderlies saw this man drunk on the 29th of February.

*Post-mortem* (five hours after death).—The membranes of the brain were intensely congested. The lungs were congested. Heart: hypertrophy of the left ventricle. Liver congested. Kidneys normal.

*Brain*.—It was in the brain only that parasites were found in any number. In other parts of the body they were scarcely to be found at all. In the brain many of the capillaries of the grey substance were filled with corpuscles, and in many of those examined there was a parasite in every red corpuscle. Many parasites were also found in some of the veins of the smallest calibre. The parasites were found almost exclusively in one stage. The patient had therefore died very shortly after a fresh generation of parasites had been set free, and had attached themselves to the red corpuscles, as they were seen as minute round bodies in the red corpuscles, of which they only filled a very small part. Parasites in any other stage were extremely few. The blood-vessels in the pia mater contained many white corpuscles, and a considerable proportion of these contained pigment, which was the chief evidence of a previous generation having existed. Whilst in many of the blood-vessels the parasites were exclusively in the early stage, in some vessels a mixture was found of very early parasites, and some in the sporulating stage with central pigment, indicating that the stage of development of the latest generation of the parasites which existed when the patient died was not exactly the same in all the blood-vessels of the brain, although it was approximately so.

*Spleen*.—Pigment was found in large white corpuscles

scattered throughout the stroma, but compared with some of the cases it was not very abundant. There was a good deal of it in the small veins. In the lymphoid tissue of the spleen there was scarcely any pigment at all, and there was none in the Malpighian corpuscles. Only a very few small parasites were seen in the red corpuscles, and these were in the same stage as the most of those which were found in the capillaries of the brain. Their number was so small that whole fields of the microscope required to be searched before one was found, and they were apparently not so abundant as Surgeon-Captain Duggan's description would have led us to suppose would have been found from the blood in the finger in similar cases.

*Liver.*—The liver cells were for the most part apparently healthy, but in a few parts there was a zone of degenerated liver cells, indicated by vacuoles, and breaking down near the central vein of the lobules. These areas of degenerated liver cells were comparatively small, as compared with the apparently normal parts of the lobules. Although in certain parts no pigment was present, in other parts there was a moderate amount of pigment, the pigment being found in white corpuscles in the capillaries and in the endothelium of the capillary wall, and very sparsely in the liver cells themselves. Most of it was found in the endothelium of the capillaries. No parasites were observed in the red corpuscles in the liver.

CASE 3 was a fatal case of pericarditis, and the tissues were examined in order to ascertain the condition of the organs in fatal cases at Sierra Leone, in which malarial fever was not recognised as present. A careful examination of the brain, spleen, liver, and kidneys failed to show a single parasite or any pigment. The man had resided two years on the West Coast before he died.

CASE 4 was a case of fatal pernicious malaria which occurred in the military hospital. The tissues were sent

by Surgeon-Captain Duggan, who obtained them at the hospital when the post-mortem was made, but he has been unable to give me any detailed clinical account of the case. His recollection is that the symptoms did not differ essentially from those that characterised other fatal cases of fever at Sierra Leone.

*Brain.*—The number of parasites found in this case was not so great as in the other cases examined, but those observed were again all found in the brain. In the sections examined the large number of capillaries blocked with parasites found in the other cases were not observed, but still a certain number of capillaries of very small calibre were observed to contain red corpuscles infected by parasites in an early stage. Amongst the red corpuscles in the veins a very small percentage were observed to contain parasites well stained and distinct, and in size slightly exceeding that observed in the earliest stage of parasitic infection, but none of them had reached the size characteristic of the sporulating stage, and they contained no pigment. In the sections examined most of the capillaries contained very little blood.

*Spleen.*—There was pigment in the large white corpuscles in the pulp. The pigment in these corpuscles was observed in two forms. In some cases it was in the form of very fine needles; in others in rounded homogeneous spheres, and in some corpuscles there seemed a transition from the one form to the other. It would seem as if the needle-shaped pigment of the sporulating parasites is very soon collected into minute spheres when taken up by the white corpuscles. Most of the pigment was contained in the pulp. There was very little in the lymphoid tissue, but a good deal was accumulated around the Malpighian corpuscles, and streaks of rounded pigment were found in the walls of the vessels. In the spleen the pigment was observed to be accumulated in blocks in some parts, these blocks being found around the Malpighian bodies (none being found inside these structures), and a long streak of such blocks was found



on the border of a tract of fibrous tissue. Although I was not able to detect the walls of the lymphatic vessels, the position of the pigment and its arrangement suggested that, in this case, the pigment in the spleen was to some extent being taken up by the lymphatics after the manner described by Bignami. No parasites were found in the red blood-corpuscles, either in the splenic pulp or in the veins, so there must have been very few of them in the spleen.

*Liver.*—Parasites were not found in the red corpuscles in the sections examined. The liver cells were, on the whole, fairly normal. The endothelium of the walls of the capillary blood-vessels contained much pigment, the endothelial cells being frequently swollen and degenerated. The degenerated cells stained deeply in cochineal. The swollen capillaries contained also white corpuscles in which there were often deposited small spheres of pigment. Occasionally a shapeless block or mass of pigment is to be seen outside the capillary vessels. In some parts of the sections pigment was found in the liver cells, mostly in the form of small round spheres, but in many parts the liver cells were free from pigment. In the interlobular vascular tracts large shapeless blocks of pigment are to be found, mostly outside the sheaths of the blood-vessels, and therefore presumably in lymphatic vessels, although no such vessels could be distinctly made out. The pigment in the interlobular tracts is more pronounced than in the lobules, as it is no longer scattered in the cells, but accumulated in blocks.

CASE 5.—The tissues in this case were obtained from the patient whose case is described by Surgeon-Captain Duggan in a paper read before the Society last session. It is Case 7 of that paper.

*Brain.*—In this case the brain was found to contain many parasites, vessels being frequently filled with blood-corpuscles, every one of which was infected. Examples of several stages of development of the parasite

were found, but very frequently capillary vessels contained parasites in only one stage. For example, in many of the vessels the small round parasite with only one nucleolar element was found occupying a small part of the red corpuscle, whilst in other vessels parasites had for the most part reached the stage in which sporulation was beginning, and in others the central pigment with the occasional evidence of distinct spores showed that the sporulating stage had been reached. Specimens stained deeply in methylene blue showed the young form of the parasite as a minute spherical body with one deeply stained nucleolar point. The later stage showed, when the staining was sufficiently successful, three to four nucleolar bodies.

On account of imperfect preservation it was difficult to say what was the number of spores which might be considered characteristic. Three and four were usually observed. In one instance six spores were counted, and were very distinct, occupying more than two thirds of the sphere of the parasite. Supposing them to have remained in their place, and that the other spores had occupied the vacant place, and had been removed in the cutting of the section, that would imply that in this particular sporulating body there might have been eight or nine spores. Yet, making allowance for the possibilities of some of the spores having been cut off in preparing the sections, and for deficiencies in staining, the number of instances in which only three and four spores were observed was comparatively large enough to justify me in assuming that some at least of the parasites had not produced more than six to eight.

The brain was the seat of small hæmorrhages. The area of free red corpuscles lying in the brain substance was not always observed in connection with a blood-vessel, but this was probably due to an accident of the plane of section, because in most cases the blood-vessel from which the corpuscles had escaped was found in their midst, and in one or two cases I was able to detect the breach or

breaking down in the vascular wall through which the blood had escaped.

The young parasites observed in the blood-vessels of the brain in this case are similar to those described by Surgeon-Captain Duggan as having been observed in the blood from the finger taken before the patient died.

*Spleen.*—The spleen contained a considerable amount of pigment, which was observed in large white cells scattered through the pulp, the veins of which particularly contained many pigment-bearing white corpuscles. The lymphoid tissue contained only a few pigmented cells; none were observed in the Malpighian follicles. The pigment in the white cells in the spleen in this case is mostly needle-shaped, but has already begun to take the form of minute spheres. In some white corpuscles the part of the protoplasm immediately surrounding the nucleus was entirely free from pigment, whilst the peripheral part of the corpuscle was filled with needle-shaped pigment in crystalline form. Tracts of pigmented white cells were observed in the course of the veins external to the vascular wall,—probably, therefore, in lymphatic vessels, or in the perivascular spaces. A very few parasites in the early stage were observed in the red corpuscles in the spleen. These were so isolated and were so few in number that frequently a whole field of the microscope would be searched without finding one.

*Liver.*—The chief feature in sections of the liver was the great distension of all the capillary blood-vessels, which were filled with blood and enormously dilated. A very few liver cells were in a state of fatty degeneration, but the most of them appeared to be healthy. White corpuscles containing pigment were found in the blood-vessels. Many endothelial cells of the vessels contained pigment spheres, and were swollen and in various stages of degeneration, and differed from the uninfected endothelial cells by taking on deeply the cochineal stain. A few liver cells contained pigment, and those of them that contained pigment were in some cases slightly changed in

appearance, and seemed to be less sound than the other cells.

If we compare the description of the first case of this disease, which was published in the seventy-ninth volume of the 'Transactions,' and the three cases which I have now described, it is at once evident that we are dealing with the same parasite in each case, and that the changes which it causes in the tissues are uniform. In the case of pericarditis, without any history of malarial fever, which was carefully examined, in none of the organs was there found any parasite or pigment, or any change suggestive of parasitic infection. As the man had been resident at Sierra Leone for two years the result is so far interesting, as it shows that residence there does not necessarily imply infection by the parasite of malaria of that coast. Dealing, then, with the four cases which were fatal by malarial fever, we find the following facts regarding the parasite. The habitat of the parasite in these cases is the brain, whilst the circulating blood, as observed in the blood-vessels of the other organs, contains only an extremely small number of isolated parasites, not even so many as Surgeon-Captain Duggan found in blood taken from the finger. The capillaries in the brain were in three of the cases filled with red corpuscles in which sometimes every one, and frequently a great number contained parasites.

In the fourth case the capillary blood-vessels of the part of the brain which was examined were mostly empty, and it consequently was very difficult to find the parasites in them, but a certain number were found. In all the cases a small proportion of infected red corpuscles were found in the veins, but the number of infected corpuscles in the veins was comparatively few. In Case No. 4, in which fewer parasites were found in the capillaries, comparatively more were found in the veins. In all the cases white corpuscles containing pigment were found in the veins, and especially in the veins of the pia mater. Pigment in the endothelium of the brain capil-

laries was more or less prevalent, but in far less proportion than what was found in the capillaries of the spleen or liver. In two of the cases the brain was the subject of small hæmorrhages.

The parasite in the four cases presented the same appearance and size in its early stage, when it is seen as a small rounded or ovoid body, of which the diameter is not more than a fifth or fourth of that of the red corpuscles. In this early stage, when stained with hæmatoxylin, it is seen as a very minute circular body, and this stained body probably represents only the nucleolar element. When successfully stained by methylene blue it is seen to consist of a deeply stained nucleolar element, surrounded by a zone of more lightly stained protoplasm, the nucleolar element being often placed eccentrically.

In the next stage it has become larger, but still only occupying a small part of the red corpuscle, and shows an appearance of spores, which, in my preparations, were usually indistinct. In the third stage, in which there is central pigment with a spore formation surrounding it, the size of the parasite is still comparatively small, its diameter not exceeding a third or a half of that of a red corpuscle. It was only in one of the cases, Case 5, that the preservation of the tissues was sufficiently good to enable the spores to be seen. (It is well known that in order to get good preparations of the parasite of malaria the tissue must be fixed and hardened very shortly after death.) For the most part, even in this case, the spores were either not visible or were too indistinct to be counted, the part of the parasite surrounding the central pigment being seen as a narrow stained zone without distinct elements. But occasionally I obtained preparations in which spores were distinct.

I have already given my reasons for believing that in this case the number of spores varied from five and upwards, to as many as eight or nine. The amount of pigment formed by this minute parasite is comparatively large.

Usually one stage of the parasite prevails in one brain, and sometimes almost exclusively. In one case (Case 5) in which all the stages were observed, one stage prevailed over the others; and when two stages were visible in the same case all the parasites in one blood-vessel were often of one stage exclusively, whilst those in another vessel were exclusively in a different stage. The stage which prevailed in all the four cases was the early stage with one nucleolar element, and it is interesting to note that the death of the patient must have coincided with the prevalence of the early form of the parasite.

We may infer also that in this form of malarial fever the tendency to periodicity is associated with a certain rhythm in the development of the parasite, a fact which is well ascertained for other forms of malarial fever associated with the varieties of the parasite of malaria.

It is noteworthy that not only were no forms of the crescent body found in the tissues in these four cases, but none of the rounded forms of parasite which the descriptions of authors and my own experience have shown me to be characteristic of crescent forms in the early stage were seen.

I will not go here into the differences that exist between this parasite and that which causes the tertian and quartan fevers. My description, and the drawings which accompany this paper, ought, I think, to satisfy on that point those who are familiar with these fevers, and those who are not have only to compare this parasite with the tertians and quartans described by Golgi and other Italian writers, or with the description given of them by Dr. Marshall and myself in the seventy-ninth volume of these 'Transactions.' If, more particularly, the drawings which accompany this paper be compared with the drawings of the quartan and tertian parasite, the difference is so distinct that it cannot be overlooked. In its morphology it so closely resembles the parasite of the summer-autumn fever of Marchiafava, and of the same or similar parasite which causes the more severe fevers in the south of

Spain, that I am unable to observe any essential difference.

We have seen that the veins in the brain, and especially in the pia mater, contain large numbers of white corpuscles laden with pigment, and presumably red corpuscles containing a parasite, swallowed as it were whole, of which I have figured one example.

One stage of development usually predominated in all the four cases—the early stage. When two or three stages were observed in the same case all the parasites in one vessel would be of one stage, and in another vessel all or nearly all in another stage, one capillary being filled with the young parasite, and another capillary containing many sporulating forms.

The walls of the blood-vessels that contain parasites sometimes break down, leading to capillary hæmorrhages.

We next find morbid appearances in the spleen. In the spleen, in all the four cases, much pigment was found, contained mostly in large white corpuscles. Very few parasites were found, so that in these cases it cannot be considered that the spleen is a habitat of the parasite, in this respect contrasting with what is known to be the case with many of the fevers that prevail in Southern Italy. Blood aspirated during life from the spleen in these four cases would not have contained the parasite in the various forms of development, such as has been described by the Italian investigators. That this negative result was not due to bad preservation of the tissue was evident from the fact that in the very few isolated infected red corpuscles that were found the parasite was sufficiently well preserved. The spleen in these cases becomes a storehouse, in which the pigment-bearing white corpuscles are collected, and these are collected almost entirely in the spleen pulp, as they are found scattered thickly through the pulp and also in the splenic veins and in the perivascular spaces.

From the spleen they find their way to the liver through the portal vein, and it was interesting to note in one

case, that while in many white corpuscles in the spleen the pigment was still in the needle form, after they had reached the branches of the portal vein the pigment had all assumed the form of small spheres.

The appearances to be described in the liver fall into two categories : first, the condition of the pigment in that organ ; and secondly, the changes observed in the tissues of the organ itself. White corpuscles containing pigment are observed in greater or less number in the capillaries according to the case, but in these cases the most of the pigment was contained in the endothelial cells of the vessels. From the endothelial cells of the capillaries some of it is seen to find its way into the perivascular spaces. When once outside the capillaries it has a tendency to accumulate in the form of blocks ; the largest of these blocks were seen (and some of them were of considerable size) in the perivascular tissue of the interlobular veins. (In connection with this description I refer to an epitome of Bignami's descriptions of the Italian fevers, which I gave in my paper of last year.) It will be seen that in all essential particulars the disposition of pigment in the spleen and liver described by Bignami holds good for these Sierra Leone cases, and the appearances were uniform in the four. It is clear, therefore, that they correspond to a general law, which may be briefly expressed by the statement that the pigment is collected in the spleen, carried to the liver, and there becomes eliminated or absorbed.

The appearances observed in the liver tissues show that this process is not carried out without a destructive effect on that organ. I pass over the small amount of fatty degeneration in the cells as not being greater than is often found in persons who have died of other diseases, but the capillary injection observed to some extent in all the cases, and to an enormous extent in Case 5, shows that the poisonous effete matters produced by the parasite have a highly irritating effect on the blood-vessels of the organ. This is made still more apparent when it is



observed that under the contact of the pigment the endothelial cells of the capillaries swell and degenerate, leading to injurious effects on the integrity of the vascular wall. The liver cells, which absorb a certain amount of the pigment, cannot fail to be injured by the contact with this foreign substance, although an apparent destruction of the cells was not observed to any great extent.

The appearances found in the liver explain the congestion and enlargement of that organ frequently found as a symptom and sequela of these fevers. It is a condition familiar to all physicians who have had much to do with the treatment of malaria. The congested and enlarged spleen, so frequent in these fevers, is readily explained in a similar way. It is impossible for an organ to be so full of foreign matters capable of exercising an injurious effect on the tissues without undergoing morbid change, but clinical observers have not hitherto associated the affection of the liver in malarial fever to the same extent with the disturbance caused by the destructive effect of pigment on the endothelium of the vessels. Surgeon-Captain Duggan informs me that the condition of the liver in the fevers of Sierra Leone is often a more important complication than that of the spleen. Whatever injurious effect is produced in the liver, as in other tissues of the body, by the poison set free by the parasite in its development, we must, in the light of the anatomical changes which I have described in these four cases, consider that a large share of the mischief is caused by the blood-vessels of that organ being charged and injured by the pigmentary refuse left by the sporulating body.

The changes in the kidney described by me in the first case were found repeated in a still more marked form in Case 2, and it is interesting to note that the destructive effects observed in the kidney epithelium were not found to be due to pigmentary accretion, or to any visible products of the parasite, and must therefore have been due to the effect produced during elimination of the intangible toxins which it generates.

It would be an interesting task to endeavour to detect the cause of the selection of the cerebral vessels by the parasite which produces these fevers, and it would not be difficult to advance various hypotheses to account for this selection; but I do not think it is desirable to burden this paper, which is mainly a record of facts, with the exposition of speculative theories.

In this connection, however, naturally the first circumstance that occurs to our consideration is the fact of the small calibre of the cerebral capillaries. Bevan Lewis remarks that, taking the capillaries of all regions, excepting the enormous capillaries of marrow, the average dimensions are between  $7\mu$  and  $10\mu$ , that is, when full of blood. The capillaries of the cortex are very often not over  $4\mu$  in diameter ( $\frac{1}{8250}$  of an inch), and they are of less calibre than the red blood-corpuscle. It follows from this fact that in the ordinary course of the circulation the blood-corpuscles must come into closer contact with the capillary wall of the cerebral vessels than in the larger capillaries of the other parts of the body, and with this close contact there is probably also associated a slow circulation.

Now we know that in these fevers in the small veins the infected red corpuscles are mostly found lying on the wall of the vessel, showing that, once they are infected, there is a tendency to their remaining adherent to the endothelium, instead of being carried on into the general circulation. The infected corpuscles can thus only be driven through the capillaries of the brain into the cerebral veins, when the *vis a tergo* is sufficient to overcome the tendency which they have to adhere to the endothelium. If this power is not sufficient, we have necessarily an arrest of the circulation of the infected corpuscles in the delicate capillaries.

Against this theory is to be placed the fact that, in tertian and quartan fevers, and in many of the cases of the fevers of the severe type associated with the parasite of the summer-autumn fevers, no such special selection of

the brain occurs. In these fevers the infected corpuscles must find their way through the cerebral capillaries without any special blocking of the calibre; and in this connection we must remember that the red corpuscle which harbours the tertian parasite becomes larger than the ordinary red corpuscle. Are we, then, to seek for the cause of the blocking of the small brain capillaries by the corpuscles infected with the parasite of these pernicious fevers, not only in the small calibre of the vessels, but in some increased toxic quality of the parasite itself, which so alters the physical properties of the red corpuscle that it adheres to the vascular wall instead of gliding on in the circulation, coupled with the fact of the short period required to produce its spores; the arrest of the circulation in the vessels and the rapidity of its reproduction leading to the capillary vessels becoming the habitat and breeding place of the parasite, comparatively few of the infected corpuscles ever passing from the brain into the veins of the general circulation? According to this theory the primary cause of the special selection of the brain is the small calibre of the cerebral capillaries in conjunction with the severe toxicity of the parasite as compared with the other forms of the parasite of malaria.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 6.)





Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.



Fig. 7.



Fig. 8.

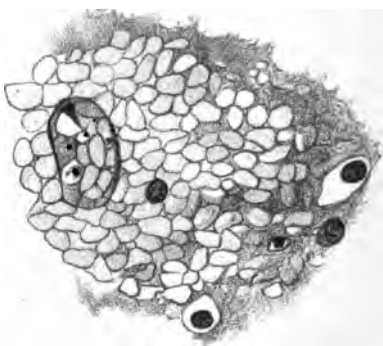


Fig. 9.

## DESCRIPTION OF PLATES I AND II

### A Further Contribution towards the Pathology of the Pernicious Malarial Fevers of Sierra Leone, and our Knowledge of the Parasite with which they are Associated (GEORGE THIN).

N.B.—All the figures were drawn as seen by a magnifying power of about 500 diameters, except Figs. 4 and 5, which represent a magnifying power of about 1000 diameters, and Fig. 10 one of 580 diameters.

#### PLATE I

FIG. 1.—A capillary vessel in the brain from Case 2 in which all the red corpuscles are infected by the parasite. The parasite is in an early stage.

FIG. 2.—Transverse section of a vein of the brain in Case 4. Three corpuscles are seen to be infected by the parasite, which is in an intermediate stage of development. In two of the infected corpuscles the lighter staining shows that the corpuscle had already begun to lose its hæmoglobin.

FIG. 3.—A red corpuscle in a capillary infected by two parasites, the size of which shows that they had already begun to develop. From Case 4. (Compare with the size in Fig. 1.) Figs. 1, 2, and 3 are stained in hæmalum and cochineal.

FIG. 4.—A blood-vessel from the brain in Case 5. Under the borax methylene blue staining the protoplasm and nuclear element of the parasite are differentiated. The parasite is in an early stage, and a minute deeply stained nucleolar point is observed in each.

FIG. 5.—Part of a vessel from the brain in Case 5 showing the formation of spores. A parasite with five spores is seen on the lower part of the drawing.

FIG. 6.—From the same brain as Figs. 4 and 5. Parasites are shown with central pigment, in one of which, at the top of the drawing, two spores are visible. In the infected red corpuscle, at the bottom of the drawing, pigment is seen with three spores.

FIG. 7.—Part of a capillary from the brain in Case 5. At the lower end a red corpuscle is recognised by its outline. It contains a sporulating parasite, in which the pigment is seen on the left and four spores on the right. The lower two spores were partly joined as if in the act of division.

FIG. 8.—Also from the brain in Case 5. Sporulating forms with the central pigment are seen in the upper part of the drawing. At the lower part a group of six spores is observed occupying little more than a semi-circle. The pigment is observed in the upper part. It is impossible to say

whether some spores had disappeared, or been in another plane of section, or whether they had been displaced. The staining had almost entirely disappeared from the red corpuscles.

FIG. 9.—From Case 5. Showing a hæmorrhage in the brain. On the left in a transverse section of a small vein the remains of parasites with central pigment are seen. The vein is surrounded by red corpuscles which have escaped into the brain substance.

## PLATE II

FIG. 10.—Part of a vein from the brain of Case 4. Two white corpuscles (*a* and *b*) are seen with pigment spheres. In *c* a white corpuscle is seen containing three spheres of pigment, and in its interior a red corpuscle, and inside the red corpuscle a parasite with a nucleolar element stained. The colourless sphere in the white corpuscle represents the red corpuscle.

FIG. 11.—From the liver in Case 4. A section of a capillary containing a white corpuscle with pigment spheres. On the left an endothelial cell is seen swollen and deeply stained.

FIG. 12.—Transverse section of a small blood-vessel in the liver in Case 4. A white corpuscle with two pigment spheres is seen inside the vessel, and two pigment spheres in the endothelium of the vascular wall.

FIG. 13.—Shows the distension of a small vessel in the liver with degenerating vascular endothelium. The endothelial cell contains two pigment spheres, and is swollen, and takes on deeply the red stain. From Case 5.

FIG. 14.—To illustrate further the degenerating endothelium in the distended capillary vessels of the liver. The endothelial cells, *a*, *b*, and *c*, are seen swollen and breaking up, taking on the red stain, and containing pigment spheres.

FIG. 15.—Shows a block of pigment which, under the microscope, could be seen to be outside the vascular wall.

FIG. 16.—A group of liver cells containing minute spheres of pigment. In the upper part of the drawing a larger block of pigment is seen lying on a liver cell between it and the degenerating wall of a capillary vessel.

FIG. 17.—Shows four liver cells to illustrate the various forms in which the pigment is observed in the cells of the liver.

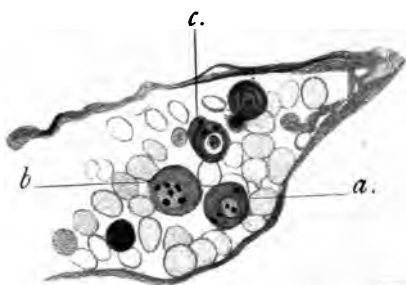


Fig. 10.



Fig. 12.



Fig. 11.

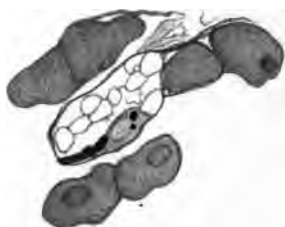


Fig. 13.

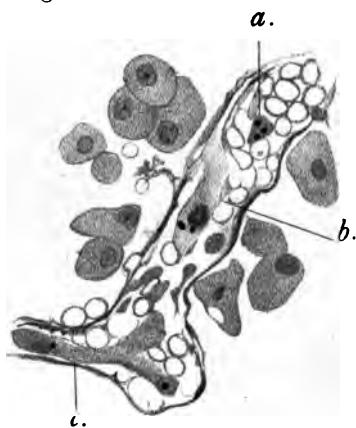


Fig. 14.



Fig. 15.

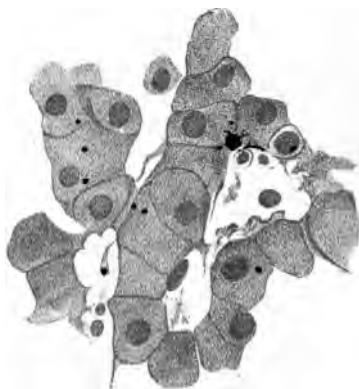
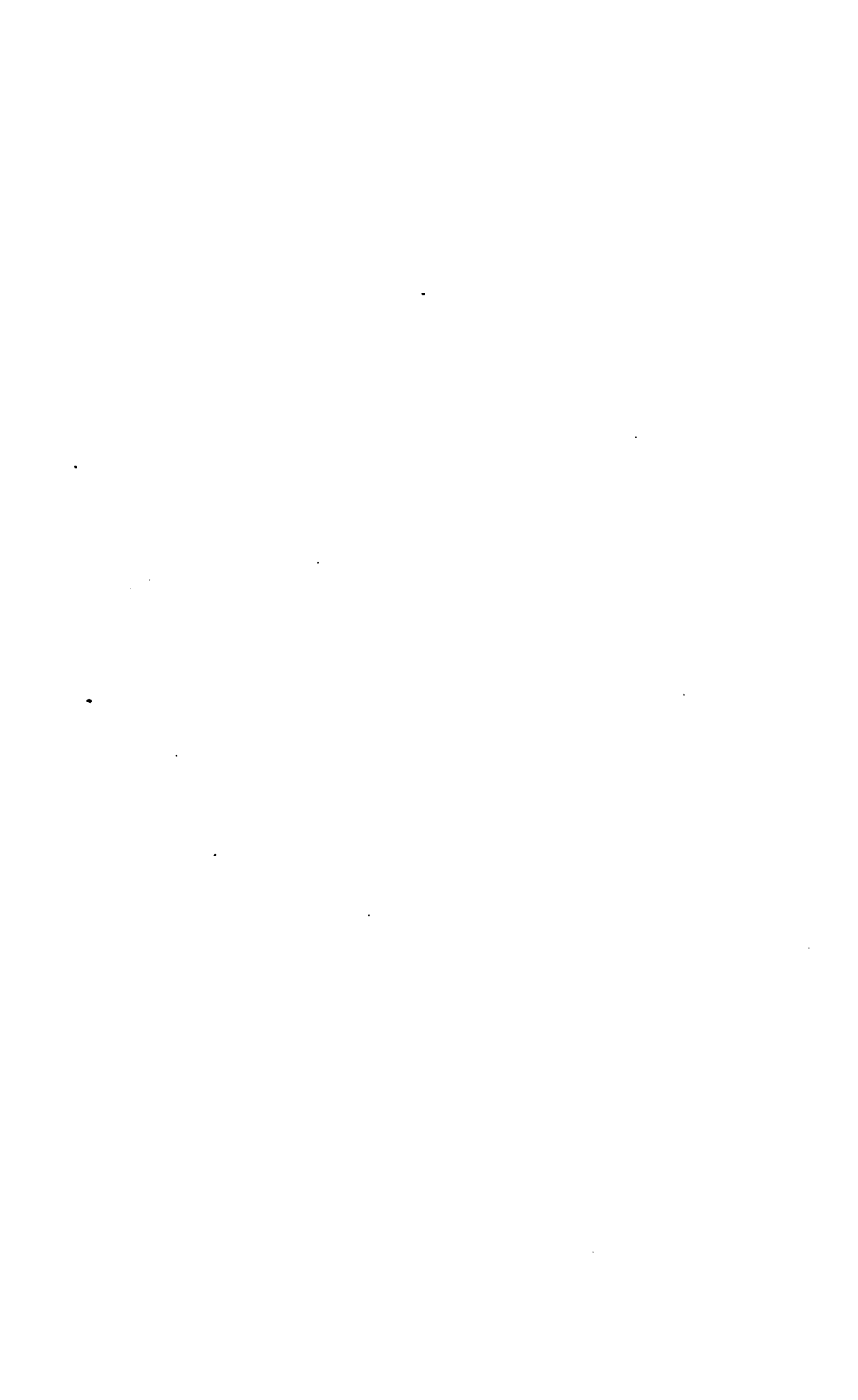


Fig. 16.



Fig. 17.





# OBSURE CYSTIC TUMOUR OF THE BLADDER

CONTAINING TWO CALCULI

SUCCESSFUL REMOVAL BY SUPRA-PUBIC  
CYSTOTOMY

BY

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THE following case is of such interest—indeed, I believe it to be unique in the annals of surgery—that I am induced to place the details thereof before the Society.

Mr. B—, an officer in the Indian Forest Department, aged 36, fourteen years resident in India, came home from Burma in the autumn of 1896, suffering from obscure urinary symptoms, which had existed ten years.

Surgeon-Colonel O'Brien, of Calcutta, under whose care the patient was in 1890, writing in May of that year, gives

such an excellent clinical picture of the case that I will quote his report verbatim :—"The patient dates the commencement of his illness to a fall in a cricket match in August, 1886. He fell on his back and was very severely shaken. Two days later he passed urine 'black as ink.' For some time after this he noticed that the urine was occasionally smoky in colour; but it was not always so, and as it did not give much trouble he paid little attention to it. Later on he consulted two surgeons in Bombay. They sounded the bladder but found nothing. He was treated with rest, citrate of potash, and aperients.

"Occasionally afterwards he used to have some difficulty in passing water. This was usually relieved by citrate of potash mixture. He got worse in Burma, but always derived benefit from the citrate of potash. In November, 1889, after getting wet snipe shooting, he noticed blood in the urine 'at the end of passing water.' He got better of this before going into the jungles for the working season, but in January, 1890, he again got very bad, passing blood every time he urinated. He returned to the station for treatment, and in ten days was so much better that he insisted on returning into camp. He was obliged to come back owing to the 'agonies' experienced in passing water. He then went to Rangoon, and was in hospital there for a week. The civil surgeon advised him to go to the sanitarium at Darjeeling.

"He was admitted into the sanitarium, and came under my care March 13th, 1890. I found him in a very weakly, blanched, cachectic condition. The daily temperature ranged from 101° to 104° F. The tongue was dry and brown in the centre. The pulse was quick (84 to 98), small and compressible.

"The urine was the colour of pea soup, and thick. On being allowed to stand in a vessel, the usual thick ropy sediment indicative of cystitis was seen. The sediment under the microscope was found to consist

almost entirely of pus. There was no alkaline condition of the urine ; it was faintly acid.

“The patient was kept in bed for three weeks, and the bladder washed daily with boric acid solution. Internally, benzoic acid, quinine, and iron were given. Under this treatment the urine gradually cleared, and the quantity of pus was reduced to a light sediment, a quarter of an inch in depth at the bottom of a 20-ounce jar of the urine. An important point is that in the normal state of the patient when free from fever, &c., the urine is always of a pale straw-colour and of low specific gravity. The specific gravity during the six weeks he was under my treatment varied between 1008 and 1012.

“The day after his arrival I got him to pass water in my presence. With great straining he got out about 2 ounces of the pea-coloured urine. I then passed without the slightest trouble a No. 8 soft catheter, and drew off 16 ounces. This occurred every time he passed urine ; he was unable to empty his bladder. I had the catheter passed three or four times a day, so as to empty the viscus completely and enable it to regain its tone. Under this treatment he recovered so far that he could pass 12 ounces at a time, but there was always an ounce or two left behind. There was never extreme pain from the stoppage during the flow, not such as would have occurred if it had been due to stone. I nevertheless sounded him carefully on four occasions, but could not discover a stone.

“He improved a good deal and gained weight in the sanitarium, but he was thrown back from time to time by intercurrent attacks of malarial fever, accompanied by the usual rigors, high temperature, and profuse sweats, ending in recovery in a few days.

“A point that struck me in connection with this case was this:—For a few days at a time the urine would show daily improvement ; then all of a sudden, and without apparent cause, half an ounce or an ounce of almost

pure pus would come away mixed with it, and settle down on being allowed to stand.

"Another point that I frequently noticed was that the urine when drawn off by a soft catheter, with patient in the recumbent position, was always clearer than that passed by the patient in the erect position and with some straining. In fact, it would appear to be clear that there is an abscess cavity in the urinary tract somewhere above the bladder (ureter or kidney). When all traces of cystitis and bladder trouble had disappeared, pus in large quantity would occasionally appear in the urine.

"Pain and tenderness were often felt and complained of in the region of the right kidney.

"The obstruction to the complete emptying of the bladder does not appear to be due to either a stone or a growth of any kind, so I think an exploration of the bladder or of the right kidney would be desirable."

Surgeon-Major Evans, under whose care the patient subsequently passed, gives a similar description of the symptoms, and came to a somewhat similar conclusion, viz. that the case was one of encysted stone or suppurative pyelitis. He further urged that the bladder should be explored by a cystotomy.

I first saw the patient on September 21st, 1896, and found that the symptoms already so graphically described, continued without any abatement. When he tried to pass water an ounce or two came away, and then the flow stopped, to go on again after a while and again stop; but he could only empty his bladder completely by lying down or by passing a catheter. He had not passed any blood since 1890. He had no pain latterly in the bladder or kidney, even when he took exercise; but when he got a jar he had some pain in the right kidney. Urine acid, sp. gr. 1022, contained much pus, but no blood or casts.

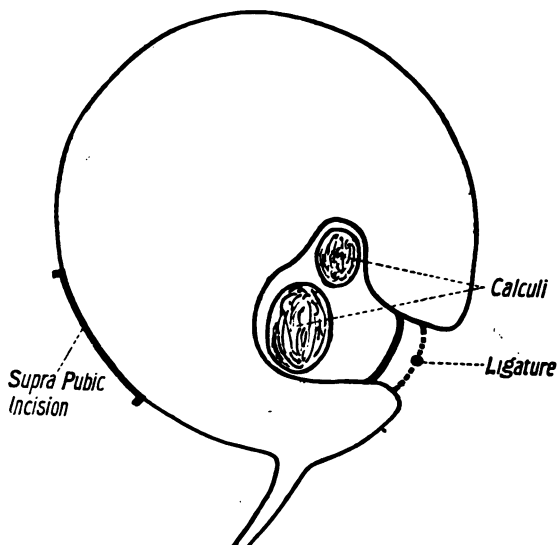
I carefully sounded the bladder both by the ordinary sounds and by the aspirator and cannula, with negative results. I therefore decided to make a cystoscopic

examination. I may say, however, that I had a strong suspicion that there was a tumour of the bladder of some kind, which fell up against the neck and obstructed the passage of water. I came to this conclusion owing to the fact that seven years ago, in a patient suffering from this particular symptom, on making a digital exploration of the bladder through the perinæum after the method then so strongly advocated by Sir Henry Thompson, I found a pendulous fibroid tumour growing from the roof of the bladder an inch from the neck, which flapped against the inner orifice of the urethra and blocked the passage. This tumour I twisted off by forceps, the patient making a thorough recovery.

On September 24th the patient entered a surgical home, and next day, Dr. Dudley Buxton giving an anæsthetic, I made a cystoscopic examination, when I at once discovered a tumour the size of a walnut growing from the lower right posterior aspect of the bladder, smooth and glistening on its surface, and attached by a broad short pedicle. There was no bleeding during the examination, the boric acid solution being drawn off as clear as when injected.

The patient left next day, and returned to the home on September 29th for operation. Next day, Mr. Carter Braine giving the anæsthetic and Mr. Cyril Wace assisting me, I opened the bladder supra-pubically, Petersen's bag being used in the rectum. On passing my finger into the bladder through the wound I at once felt the tumour projecting from the right side of the base of the bladder, and attached by a smooth, tense, round pedicle  $\frac{3}{4}$  inch in diameter. The tumour was more or less bilobed, and had a small papillomatous growth on its surface. The forceps was introduced, the tumour caught and twisted, when a small portion including the papillomatous growth came away. I then grasped the main body of the tumour and drew it into the wound. It looked like a large strawberry mass, and felt quite hard to the touch; and on scraping it with the finger-

nail, to my astonishment it was found to contain a uric acid stone the size of a nutmeg (weight 29 grains),



which was shelled out by the finger-nail and pressure of the forceps. On letting go the tumour and passing the finger into the bladder, a second stone the size of a large pea (weight 12 grains) was found. The tumour now felt flaccid and empty, like the finger of a glove. In addition to the calculi it had evidently contained fluid. I grasped it by the forceps, withdrew it through the wound, clamped the base with another forceps, and cut off the main portion of the cyst. The remainder was burnt down to the clamping forceps by Paquelin's cautery. As the lumen of the pedicle when compressed by the forceps was about an inch long, and not knowing the nature of the cyst or what cavity it might communicate with, I passed a needle armed with a double silk ligature through the pedicle behind the forceps, and tied it in two parts. Fearing to cut off the ligatures short lest a phosphatic concretion might form on their ends, I withdrew them through the

wound, inserted a half-inch drainage-tube, and dressed the wound as usual. The ligatures came away on the tenth and twelfth days respectively. The patient made an uninterrupted recovery. Urine began to pass through the urethra on October 13th, the patient began to sit up on the 20th, and the abdominal wound was completely closed on November 3rd. The patient went home on November 10th. On the 24th of that month he wrote, "I am perfectly well in all respects; I never think of my waterworks now." I saw him on March 1st, this year (1897); he was in perfect health, and had been playing hockey in a public match the day before.

This case presents many points of interest. 1, and most important, what were the origin and nature of the tumour? and how did the calculi get inside it? I may at once say that microscopically the tumour is found to be a thin single-walled cyst, composed of fibrous and muscular tissue, and covered on both the outer and inner surfaces by transitional epithelium, such as is found in the bladder and ureters. The small papillomatous growth torn from its surface is composed of dense fibrous tissue covered by the same kind of epithelium. Small non-bleeding papillomata of this kind are not unfrequently seen during cystoscopic examination growing from the bladder wall, particularly in the vicinity of the ureteral opening, and are, as a rule, harmless. The cyst wall being single precludes the possibility which might suggest itself of the two calculi having got encysted in the ordinary way in an outward pouch of the bladder, and then this outward pouch with its contents getting retro-flexed on itself, turning inwards and carrying a second layer of the bladder wall before it.

The possibility of the tumour consisting of a prolapse of the ureter through the ureteral opening also suggested itself to my mind, and, I may tell you, gave me some anxiety during the first few days after the operation, lest I had ligatured the lumen of the ureter in its entirety. I consoled myself, however, with the reflection



that no trace of a ureteral opening was found in that portion of the cyst cut off, that no symptoms such as would occur had the ureter been completely obstructed supervened, and that had any such symptoms set in I could remove the ligatures without difficulty.

The most reasonable theory to my mind is this: that the calculi passed down from the kidney as far as that portion of the ureter which passes obliquely through the bladder wall before ending in the ureteral opening, and getting impacted there, and impelled by the force of the flow of urine from behind, bulged the ureteral wall, here covered only by mucous membrane of the bladder, into that viscus. Thus eventually a pendulous cystic tumour containing the two calculi, and rendered tense by the pressure of urine from behind, was formed, projecting into the bladder, and presenting the appearance on cystoscopic examination of a fibroma.

The origin of the pus was probably a pyelitis resulting from the irritation caused by obstruction of the ureter. The pus probably accumulated in the cyst and in the dilated ureter above it, and flowed intermittently through a patent ureteral opening. In the operation this sac was cut off, the ureteral wall repaired, and the ureteral opening left untouched and patent.

Whether the injury sustained in the cricket-field had anything to do with the formation of the calculi is doubtful. Possibly one or both were present in the kidney at the time, and shaken out into its pelvis by the fall.

2. The case presents an excellent illustration of the great utility of the judicious employment of the electric cystoscope for diagnostic purposes in obscure bladder disease.

3. I am not aware of any case on record where a ligature has previously been applied to the pedicle of a tumour of the bladder. The only untoward symptom that ensued from this was that the ligatures were covered by slimy and bloody mucus, and I have no doubt that

their presence retarded by a few days the closure of the abdominal wound. I shall have no hesitation in future in ligaturing the pedicle of a bladder or prostatic tumour, when, to prevent hæmorrhage or for other reasons, it may seem desirable to do so, bringing out the ligatures through the supra-pubic wound, so that eventually they may come away in their entirety.

#### ADDENDUM.

I saw Mr. B— again on September 30th, 1897—the anniversary of the operation. He was in perfect health. about to return to duty in India, and a week before had been accepted by an insurance company as a first-class life.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 15.)



# ON MESENTERIC CYSTS

WITH

TWO CASES IN YOUNG CHILDREN SUBJECTED TO  
OPERATION

BY

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Received April 14th—Read November 9th, 1897

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PERCY M—, aged 11 weeks, was admitted to the Evelina Hospital April 23rd, 1896.

*History.*—The mother stated that the baby was plump at birth, and was brought up on cow's milk and barley water. He had gradually wasted since birth, and from the age of three weeks had not passed solid motions, only dark offensive fluid. There had been frequent vomiting of fluid, which the notes state was fæculent.

*Condition on admission.*—A pale, marasmic child, temp. 96·8°, pulse 120, resp. 40; organs natural. Abdomen rather full. Occupying the right side of the abdomen, a little below the level of the umbilicus, was a well-defined, smooth, rounded, fluctuating swelling the size

of a small kidney. It could be moved very freely all over the abdominal cavity. The swelling could be felt *per rectum*, and did not appear to be connected with any of the abdominal organs. During the first day after admission the child vomited, and its motions were loose. Subsequently the vomiting ceased, and his general condition improved somewhat. A diagnosis of mesenteric cyst was suggested; and as it appeared probable that the previous abdominal disturbance was due to the tumour, an exploratory operation was determined upon.

*Operation* (May 1st).—The abdomen was opened in the median line by an incision commencing 1 inch above the umbilicus. The tumour, which occupied the right lumbar region, was drawn out, and found to be a cyst of the size of a Tangerine orange. It occupied the border of the mesentery, and a knuckle of moderately compressed intestine ran over its free surface for about 4 inches. The intestine, in fact, was incorporated in its wall. Numerous enlarged glands were visible in the mesentery. Two ounces of fluid were withdrawn by aspiration, and the puncture was closed with pressure forceps. The coil of intestine was then returned into the abdomen, and the cyst so attached to the parietal peritoneum as to leave a portion of its wall projecting externally. The angles of the wound above and below were closed.

The child was very sick after the operation, and vomiting continued until the 3rd inst. No milk was retained. Sickness was arrested by washing out the stomach. Raw meat juice and nutrient enemata were substituted for the milk. On the morning of the 3rd inst. he was transfused with a pint of saline solution.

May 8th.—No vomiting had occurred since the 3rd inst. Under an anæsthetic the exposed portion of the cyst wall was incised, when the fluid was found to have re-accumulated. The fluid let out was of a reddish-brown colour, odourless, and towards the end of the drainage some solid material of a green and yellow colour came away. The cyst was washed out and a drainage-tube

inserted. No suppuration occurred either in the wound or the cyst. The child's general condition improved, all sickness ceased, and the cyst discharged only a little clear fluid.

He was quite convalescent and continued well until June 5th, when he vomited twice. Next day there was no sickness, but the breathing became bad at 7.50 p.m. At 8.20 the child became convulsed on both sides, the eyes twitching. The pulse after the convulsions was almost imperceptible. The collapse continued, and death took place at 10.30 p.m., nearly six weeks after the operation.

*Autopsy.*—The peritoneum was perfectly healthy. The cyst lay between the layers of the mesentery of the jejunum, and was intimately connected with the intestine which coursed over it (fig. 1). So close was the relation between the two that the cyst might have originated in the space where the layers of the mesentery divide and surround the intestine. The portion of intestine (about 2 inches in length) attached to the cyst, although not in the least compressed by it, was distinctly smaller in calibre than elsewhere. The diameter of this part was  $\frac{3}{8}$  inch, while that immediately above was 1 inch in diameter. This narrowing had the appearance of being the result of arrest of development of the tube from long-continued pressure, and possibly interference with the blood-supply from the constriction of the vessels by pressure of the cyst. In the specimen<sup>1</sup> (fig. 2) it may be seen that where the intestine leaves the wall of the now distended remains of the cyst it is somewhat suddenly bent. The constricted or narrow part in the recent state was blocked with casein, and there is further evidence of pre-existing chronic obstruction in the dilatation of the intestine on the proximal side of the tumour. The cyst is oval in shape, and measures 2 inches in its longest diameter, which lies parallel with the intestine. Its inner surface

<sup>1</sup> Preserved in the Museum of the Royal College of Surgeons.

is smooth, and its walls firm and 3 lines in thickness. Under the microscope (see figs. 3 and 4) it showed no endo- or epithelial lining. Just beneath the surface was a thin layer of unstriped muscle-fibre cut longitudinally, then a thick layer of muscle divided transversely, followed again by another thick layer composed of several fasciculi running parallel to the surface. The contained fluid was turbid serum, in which were white and red corpuscles, with no fatty corpuscles indicative of chyle. There were  $\frac{1}{10}$  of albumen. In the neighbourhood of the cyst were many slightly enlarged, but not tuberculous glands, as was proved by microscopic examination. All the abdominal organs were healthy.

It may be fairly claimed that the child had completely recovered from the operation, and that this had accomplished all that was possible. The child had, before admission, suffered with a severe convulsive seizure.

The cause of the fatal convulsion was not apparent. There was no evidence at the post-mortem of torsion or kinking of the gut.

I was indebted for this case to my colleague, Dr. G. Carpenter, to whom it was sent by Dr. R. G. Reid; Dr. Carpenter also suggested the diagnosis of mesenteric cyst after rectal bimanual examination under an anæsthetic.

CASE 2.—Albert G—, aged 3 years and 8 months, was admitted to the Evelina Hospital on June 27th, 1896, with intestinal obstruction. The parents stated that the child had always had a swollen belly, and was in the hospital in September, 1895, for diarrhœa and vomiting. He was discharged as cured in four days. No action of the bowels had taken place since June 23rd, four days before admission. From that date the child had vomited incessantly. He presented the typical abdominal *facies*, and was evidently much exhausted. The abdomen was much distended but flaccid. No tumour could be felt either by palpation or *per rectum*. There was dulness on

percussion in the right flank, extending to within 3 inches of the middle line. No thrill felt. Rectal injections were followed by the expulsion of a few small pellets of faecal matter.

*Operation.*—Laparotomy under A.C.E. was performed three hours after admission. On introducing the hand an ill-defined swelling was felt in the right flank; it was drawn out of the wound. This proved to be a thin-walled cyst in the free end of the mesentery. The intestine coursed over and was incorporated with its walls, being here completely empty and flattened like a riband. It was considerably distended on the proximal and empty on the distal side. The cyst was so thin-walled and flaccid, that even with the hand in the abdomen its outline could not be made out; and it could not be differentiated by the touch from distended coils of intestine. Hence the difficulty in recognising its presence except by percussion before operation.

After drawing off 32 ounces of clear straw-coloured serum, I opened the cyst with the intention of shelling it out or excising it. The latter alternative seemed possible, as the walls appeared before tapping to be entirely free from vessels. Enucleation being impossible, nearly the whole of the cyst wall was removed with the exception of the lateral margins, over which the mesenteric vessels ran. The inner surface was cleansed, and the cut edges brought together with continuous sutures of silk. The knuckle of bowel was then returned into the abdomen and the wound closed. After emptying the cyst the bowel on the distal side immediately became filled with the intestinal contents. After the operation, which was not of long duration, the child was very restless, shrieking and starting up. Pulse weak and at times imperceptible.

Enemata of coffee and brandy and hypodermic injections of strychnine were administered. Later on bromide of ammonium was given, with good results in diminishing the restlessness; but the child never rallied, and died



nine hours after the operation, whilst being transfused with saline solution.

*Autopsy.*—The organs generally were healthy. The cyst was situated 7 feet from the ileo-cæcal valve. The intestine attached to it was not constricted. There was no peritonitis or peritoneal injection. The specimen<sup>1</sup> (fig. 5) shows the remains of the cyst, which have been distended and hardened. The intestine is attached to its surface for a distance of 3 inches to 3½ inches; its proximal end is dilated, and its distal end somewhat contracted. The cyst was evidently formed by a separation of the two layers of the mesentery in immediate contact with the intestine. Its walls were extremely thin and the inner surface smooth. Under the microscope there was no lining. The inner half was composed of connective tissue containing granulation cells, and the outer half of loose connective tissue and fat; no unstriped muscle tissue. The cyst fluid was clear serum, which became solid with albumen on boiling, and contained a large quantity of cholesterine, but no fatty matter.

*Remarks.*—These two cases illustrate in a striking manner the symptoms associated with mesenteric cyst in the earlier and later stages. In the first case the prominent features were vomiting and diarrhœa with emaciation, amounting to marasmus. These dated from birth. In the absence of an abdominal tumour they would certainly have been ascribed simply to gastro-enteritis. In fact, they were due to this cause, but it resulted not from faulty feeding, but from chronic obstruction high up in the intestinal canal.

In the second case the tumour declared its presence by an attack of complete intestinal obstruction. The only premonitory symptom was the illness with vomiting and diarrhœa a year earlier.

Although it is generally assumed that a mesenteric cyst if left alone will ultimately produce obstruction by

<sup>1</sup> Preserved in the Museum of the Royal College of Surgeons.



MR. EVE, ON MESENTERIC CYSTS.

FIG. I.



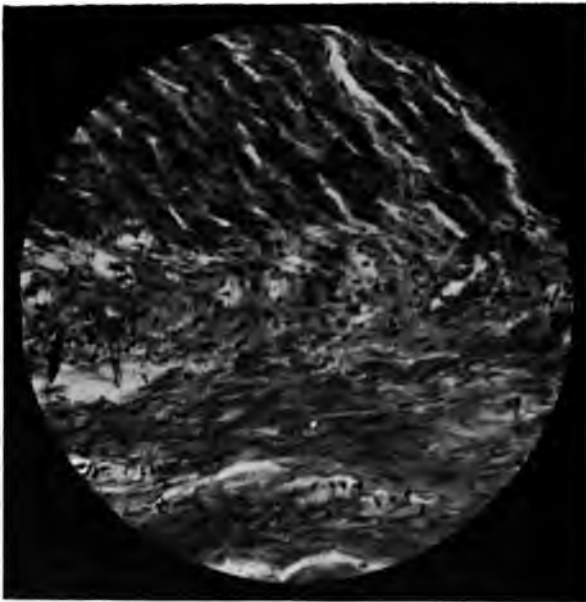
FIG. II.



Mesenteric Cyst. Case 1. The Cyst is shown after distension with spirit. A rod is placed in a sinus opening on the surface at the site of the incision.

Mesenteric Cyst. Case 1. The photograph shows the narrowing of the knuckle of intestine where in contact with the Cyst. The size of the intestine above the Cyst may be seen in Fig. I.

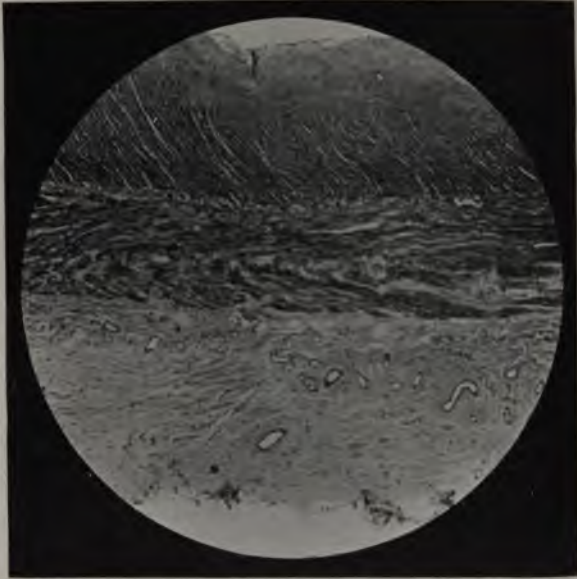
FIG. III.



Wall of Cyst from Case 1, showing bands of unstriped muscle. High power.

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FIG. IV.



Wall of Cyst from Case 1, under low power. The inner half, which is uppermost, is largely made up of unstriated muscle.

FIG. V.



Mesenteric Cyst. Case 2. Showing seat of Cyst which was excised. The sutures have been removed from the side towards the observer.



occluding the bowels, an examination of the recorded cases reveals the fact that this has rarely occurred. A specimen is preserved in the museum of the Royal College of Surgeons, No. 2352 E, from a girl aged nine, who was seized with pain in the abdomen and vomiting, which continued till her death six days later. A patient of Brentano's,<sup>1</sup> a woman aged forty-two, exhibited symptoms of internal strangulation after two attacks of peritonitis. Laparotomy revealed in the left descending mesocolon, a cyst, which was successfully enucleated.

Pain is not usually present, but in a case recently recorded by Dr. J. O'Connor<sup>2</sup> this was a prominent symptom.

*Diagnosis.*—With one exception, Case 1 presented all the physical signs characteristic of mesenteric cyst. A spherical, well-defined, distinctly fluctuating tumour was present in the usual situation, viz. on the right side of the abdomen a little below the level of the umbilicus. It was extremely mobile, and separated from the pelvis by a distinct band of resonance. It had no connection with either the liver or the spleen. But the most characteristic symptom of all, the existence of a band of resonance due to intestine passing over the otherwise non-resonant tumour, was absent, probably on account of the small size of the bowel. This symptom was also absent in the second case, on account of compression of the gut.

In Case 2 the tumour was masked by distended coils of intestine. The only indication of its presence was dulness in the right flank, which might have been due to localised peritonitis with effusion; in fact, it is generally admitted that such a condition is in many instances indistinguishable from mesenteric cyst.

It may briefly be mentioned that the conditions, between which and mesenteric cyst a differential diagnosis is most difficult, are cysts of the pancreas, floating

<sup>1</sup> 'Revue des Sciences méd.,' tome xlvii, p. 664.

<sup>2</sup> 'Brit. Med. Journ.,' Feb. 13th, 1897, p. 391.

kidney, lipoma and myxo-sarcoma of the mesentery or retro-peritoneal tissue, and ovarian, parovarian, and uterine tumours.

I saw lately in a middle-aged woman a small ovarian cyst with so long a pedicle, and so mobile, that it could be pushed all over the abdomen and into the left loin like a floating kidney; and a distinct band of resonance existed between it and the pelvis.

Hahn<sup>1</sup> and Augagneur seem to have been the first to classify mesenteric cysts. Following these authors, Braquehay<sup>2</sup>, in an exhaustive article published in 1892, classifies them as follows:

1. Sanguineous cysts. These are usually traumatic, and occur as diffuse hæmorrhages into the mesentery, into pre-existing serous cysts, or into solid tumours.

2. Lymphatic cysts, comprising chylous and most of the serous cysts. These arise in the thoracic duct, in the chyle vessels, or in the lymphatic glands of the mesentery.

3. Congenital cysts, chiefly dermoid, but occasionally cysts of the Wolffian body.

The two cases described above belong to the second variety of lymphatic cysts, and among them fall in the largest group of serous cysts. These are believed to originate in lymphatic glands, and it is stated that remains of lymph-gland tissue have been found in their walls. This could not be demonstrated in the cases related above.

The specimen already mentioned preserved in the Royal College of Surgeons resembles the cyst from Case 1 in that a considerable quantity of unstripped muscle tissue existed in its walls. It also occupies the same position in relation to the bowel. Various suggestions may be made to account for the presence of unstripped muscle. It is present normally in the mesentery; it also exists in the capsules of lymphatic glands and in

<sup>1</sup> 'Berliner klin. Woch.,' 1887.

<sup>2</sup> 'Archives gén. de Médecine,' vol. ii, 1892, p. 291.

the walls of the larger lymphatic vessels. It is, however, difficult to believe that such a large quantity of unstriped muscle could be derived from any of these sources. Seeing the close proximity of the cysts to the intestinal wall, one is tempted to suggest that they are sometimes derived by some irregularity of development from the mesoblastic portion of the intestinal tube. In discussing the origin of these cysts E. Pagenstecher's case<sup>1</sup> may be quoted. It is that of a girl aged ten, with a lymph-gland cyst of the mesentery stated to be of tubercular origin.

Contrary to all previous observations,<sup>2</sup> Heinrich records a case of cyst in the ascending mesocolon, which possessed an endothelial lining. He supposed it to have originated from the subcæcal fossa.

Braquehaye asserts that the cysts are situated in the mesentery of the small intestine, usually near the spinal border. But since the publication of his paper several cases have been reported, besides that of Heinrich's above quoted, in which the cyst occupied the mesocolon.

Brentano<sup>3</sup> records a cyst of the left iliac mesocolon.

In the museum of the Royal College of Surgeons is a specimen presented by Dr. Bantock (No. 2352 D) ; a cyst 5½ inches in diameter, which he shelled out from the transverse mesocolon behind the stomach.

The patient, a woman aged forty-five, made an excellent recovery.

*Treatment.*—The manner in which the cyst was dealt with in Case 1 is that recommended by most writers on the subject. This is often spoken of as marsupialisation. Mr. Greig Smith<sup>4</sup> with others recommended that the cyst should be laid open at the time of operation, and stitched to the parietes.

The practice of allowing the cyst wall to become

<sup>1</sup> 'Revue des Sciences méd.,' tome lxxiii, p. 241.

<sup>2</sup> Ibid.

<sup>3</sup> Ibid., tome xlvii, p. 664.

<sup>4</sup> 'Abdominal Surgery,' vol. ii, p. 1064.



adherent to the abdominal wound before opening it is perhaps safer, seeing that in many instances the cyst is in close contact with the intestinal wall, and therefore its contents are likely to be infected with micro-organisms. Its contents are, of course, drawn off immediately by aspiration.

The other method of dealing with these tumours is by excision. One of the difficulties besetting this operation is due to the distribution of the mesenteric vessels over the cyst wall. Not infrequently it is surrounded by adherent coils of intestine.

Very exceptionally the cyst can be shelled out. This was successfully done in the cases quoted above of Brentano and Bantock. Its occasional feasibility may be brought forward as an argument in favour of laying open the cyst in the first instance.

The drawbacks of marsupialisation are—

1. That the process of cure is tardy, and a fistula may persist for a long period. In a case of Sir Spencer Wells a fistula still remained at the end of a year.

2. Recurrence may take place, the fluid re-accumulating in the cyst. It is rare, only occurring once in twenty-eight cases tabulated by Braquehay, and can clearly be avoided by keeping the orifice patent for a sufficient time to allow of contraction of the cyst.

3. Yielding of the cicatrix and the formation of a ventral hernia. This accident occurred in three out of the twenty-eight cases, and an additional case has since been published by Brentano. Operation was required in three out of the four cases. Ventral hernia did not occur once in the cases treated by excision.

4. Marsupialisation is also not free from danger, owing to twisting, kinking, or torsion of the proximal side of the gut. In a case published by Rötter in 1895 the cyst, which was situated above the umbilicus, could not be extirpated. It was therefore attached to the parietes. The patient suffered with uncontrollable vomiting, and died on the sixth day.

The cyst occupied the mesentery of the jejunum 10 cm. from the duodenum, and the intestine was obstructed owing to torsion on its axis. In Löhlein's case a second laparotomy was necessary, owing to internal strangulation by adhesions between the cyst and parietes.

When I performed the operation on Case 2 I had not dissected the specimen from Case 1, and thought that kinking might have been responsible for the death of the patient. It was this which chiefly determined me to perform excision in the second case.

5. Another complication is chyloorrhagia, which may lead to rapid and marked emaciation. This was very pronounced in Dr. Rasch's case.<sup>1</sup>

The statistics of Braquehay have demonstrated that marsupialisation in point of safety is far superior to excision. The former give 93 per cent. of recoveries, the latter 60 per cent.

The above remarks regarding treatment do not apply to the third group of congenital mesenteric cyst. These are mainly dermoids, and could usually be enucleated without difficulty.

All writers are agreed that operation is absolutely necessary in all cases of mesenteric cyst, in view of the persistence and progressive nature of the symptoms. If left alone, death may take place from obstruction, rupture or perforation of the bowel, or emaciation. It may also be said that operation is usually necessary to establish the diagnosis.

Considering the large number of cases of mesenteric cyst which have been collected, chiefly by Continental writers, it is surprising that so little attention has been devoted until recently to this subject in works on surgery and its branches.

Up to 1892, 104 cases had been collected by Delmez, Arekion, and Braquehay respectively. To English surgeons it is of special interest to recall the fact that John Hunter described some cysts in the mesorectum of

<sup>1</sup> 'Trans. Obstet. Soc.,' 1889.

hogs containing air. These had been supplied to him from Somersetshire by Edward Jenner. Dr. W. H. Dickinson described in the 'Pathological Society's Transactions' for 1870-71 one of the earlier cases, a solid and cystic tumour of the mesentery. In this country also cases have occurred in the practice of Sir Spencer Wells, Bantock, Horrocks, Lawson Tait, Thornton, Rasch, and others.

The cases which have occurred in early life are few in number. My own patient (Case 1) aged three months is the youngest I have been able to find, certainly the youngest operated upon. Winiwater records an example in an infant of four months treated successfully by repeated punctures made through the skin; Ducasset one of eleven months found post mortem, and causing death by obstruction.

#### ADDENDUM.

At the request of the Council I append brief details of the method of transfusion employed in the above cases.

The apparatus employed was that supplied by Messrs. Down Bros., as arranged and described by Dr. Horrocks; but a glass syringe was used instead of a funnel. One of the veins on the front of the elbow is exposed and opened in the usual way, the veins, if necessary, having been rendered prominent by placing a ligature above the elbow.

The fluid used is sterilised water to which  $\frac{1}{2}$  per cent. (*i.e.* one teaspoonful to the pint) of common salt has been added to increase the specific gravity. The water is sterilised by boiling, and its temperature is lowered to 102° F. either by adding cold water, previously sterilised; or by allowing a jug of boiling water to stand in a basin of cold water constantly replenished, until the temperature is sufficiently lowered.

In performing the injection it need hardly be said that precautions are taken against the entrance of air. The

tube and cannula are filled with salt solution before inserting the latter ; and the syringe is constantly kept above the level of the cannula.

The speed at which the injection is carried out is of no importance.

The amount necessary must be gauged by the condition of the pulse, which rapidly improves as the injection is continued. The minimum quantity even for very young children may be placed at about a pint.

I have never found any difficulty in transfusing very young children. The age of the patient whose case is described in the foregoing paper was 11 weeks. Quite recently I transfused a child of four months for collapse supervening after excision of the knee-joint, performed on account of suppurative arthritis following the bursting of a tuberculous osseous lesion. Before the injection the child was pulseless; it quickly rallied, and subsequently progressed favorably.

In severe cases of shock and collapse in young children, one or two teaspoonfuls of brandy may advantageously be added to the salt solution. Lately, after amputating the thigh at the hip-joint for a "railway smash" in a young man, I injected into the femoral vein one ounce of brandy with five to six pints of salt solution. Meanwhile my house surgeon amputated, at the ankle, the opposite foot, which was also crushed. The man was quite pulseless before the injection. He quickly rallied and made a speedy recovery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 20.)



# DISCUSSION ON THE PREVENTION OF ENTERIC FEVER,

(*November 23rd, December 14th, 1897, and January 11th, 1898*)

OPENED BY

GEORGE VIVIAN POORE, M.D.

AND CONTINUED BY

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## POLLUTION OF PUBLIC SUPPLIES.

THE Registrar-General's returns show that the death-rate from enteric fever, which was 322 in 1871-80, had fallen to 200 in 1881-90.

Our satisfaction at this decline is seriously qualified by the fact that the tendency of towns to suffer from epidemics of the disease seems to be as great if not greater than ever. At present there are three towns (Maidstone, King's Lynn, and Ligoniel, a suburb of Belfast) in which enteric exists in epidemic form as a result of the pollution of a public water-supply. Maidstone has had an amount of enteric fever in the last

three months which, under ordinary circumstances, should have been spread over twenty-five or thirty years, and it is evident that this is a calamity of a kind which is not compensated by a decline in the endemic death-rate. I have collected for the purpose of this discussion short records of forty-six instances in which pollution of public water-supplies has taken place, resulting in outbreaks of enteric fever more or less severe. In thirty of these outbreaks the numbers of persons who suffered is given, and I find they amount to 16,576. I am, therefore, well within the mark when I say that for the last thirty years considerably more than 500 persons per annum have suffered from enteric fever as a result of the pollution of public water-supplies.

I am mainly indebted for my information to the reports made to the chief medical officer by the inspectors of the Local Government Board. These reports are among the *chef d'œuvres* of English medical literature, and one hopes that they may be at some time re-edited and made more available, than at present is the case, to the great bulk of the profession. For six of the cases which I have tabulated I am indebted to the 'Tabular Analysis of Water-borne Typhoid,' lately published by Mr. Ernest Hart.

The contagion of enteric fever is contained in the excreta of the patient (certainly in the fæces, probably in the urine), and it is conveyed to the healthy person in his drink or in his food, and probably in the air he breathes. When enteric fever has occurred in epidemic form, the poison has usually been conveyed by water, often directly, and only less often indirectly, through milk or other beverages. In order to keep my remarks within bounds I shall not further allude to milk epidemics.

The following is a brief tabular statement of the Contamination of Public Water-supplies, 1867-97 :

Date.	Name of town.	Source of supply.	Where polluted.	Number of persons attacked.
1867	Guildford	Chalk well	Source and course	250
1870	Warwick	?	?	—
1873	Sherborne	Wells	Periphery	243
1874	Lewes	?	Course	486
"	Hull	?	"	526
"	Over Darwen	"	"	2035
1876	Tideswell	"	"	—
1878	Dewsbury	"	Source and course	600
1879	Okehampton	"	Periphery	—
"	Swansea	Upland	Source	640
"	Caterham, &c.	Chalk wells	"	352
1880	Enfield	?	Periphery	—
"	Haverfordwest	"	Course and periphery	—
"	Sandown	Stream	Source	—
"	Ventnor	?	Course	—
"	Llanely	Stream	Source	—
"	Perth	River	Course	162
"	Newlyn	Well	Source	—
1881	Blackburn	—	Course	238
"	Bodmin	—	Periphery	—
1882	Bangor	—	Course	548
1883	Hitchin	Well	Source	—
1884	Sheerness	—	Course	—
"	Kidderminster	Well	Course	1200
"	Beverley	Chalk well	Source	231
1885	Faldingworth	Well	"	—
"	Fareham	—	Course and periphery	150
"	Swansea	Upland	Source	549
1887	Margate	Well	"	—
1887-8	Mountain Ash	—	Course and periphery	518
1888	Buckingham	—	Course	—
1889	Longton	—	?	155
"	Rochester	—	Periphery	—
"	Houghton-le-Spring	Well	Source	258
1890-93	Newark	River	"	297
1890-91	Tees Valley	"	"	1463
1891	Rotherham	Streams	"	211
1891-2	King's Lynn	"	"	226
1892-3	Chester-le-Street	"	"	58
1893	Atherstone	?	Course	112
"	Paisley	Upland	Source	672
"	Worthing, &c.	Wells	Source and course	1411
1894	Newport	"	Source	516
1895	Raunds	"	"	129
1897	Maidstone	Springs	?	1890 ?
"	King's Lynn	River	?	450 ?



Of these 46 instances of pollution of a public water-supply it will be noted that in 3 cases the water was polluted both at its source and in its course, and in 3 cases in its course and at the periphery. In 18 cases it was polluted at its source alone; in 13 cases it was polluted in its course alone; in 5 cases it was polluted at its periphery alone; in 4 cases the fact is not stated.

#### POLLUTION AT THE SOURCE.

Of the 21 instances in which pollution occurred at the source it was shown that of such polluted sources 11 were wells, 7 were streams and rivers, and 3 were upland sources.

These streams and rivers had been polluted by the water-carried excrement flowing into them through the drains and sewers of places higher up, and in some few instances by the falling in of excremental matters from the banks or from boats and barges. Every river and stream in the country is more or less polluted in one or other of these fashions. This fact is too familiar to need further comment.

The mode of pollution of the wells is of more importance.

Three of these polluted wells are of a common type, *e. g.* at Newlyn, Faldingworth, and Raunds, where the wells were polluted quite in the ordinary domestic way by the leakage into them, from neighbouring drains, of foul liquid, either from closets or washhouses where linen fouled by enteric had been washed. In the cases of Guildford, Caterham, Beverley, Hitchin, Margate, Worthing, and Newport we have instances of the fouling of deep wells in the chalk. A deep chalk well is generally considered a safe source for water, and some appear to think that safety is proportionate to depth. It seems necessary to say that depth is no protection against the effects of leakage at any point, for leakings must fall to the bottom. It is necessary also to say that the difficulties of inspection

are very largely proportionate to the depth. Again, as leakage may occur at any point in the walls, the area of the walls is in some sense a measure of the vulnerability of the well. The chemist and bacteriologist may report with regard to a particular sample taken at a particular time that the water is "of excellent dietetic quality, well suited for the supply of a town," but the merest tyro in well-sinking might point out the dangerous position and formation of the well.

With regard to these eight wells the following particulars may be given.

1. At *Guildford* the pollution of a new chalk well was caused by the leakage into it of quantities of sewage from an old ill-constructed sewer.

2. At *Caterham* an adit over 400 feet below the surface was definitely fouled by a workman suffering from enteric, who deposited his evacuations in the tunnel. The investigation of this classic case was carried out with remarkable acumen by Sir Richard Thorne. The following are the facts:

1879. *Red Hill and Caterham* (Dr. Thorne Thorne).—352 cases caused by a labourer who was working in the adit of the water company, and who was suffering from enteric at the time, actually depositing his evacuations in the tunnel. The evidence shows that he began to be ill on January 5th, and that he continued to work in the adit until the evening of January 20th, when he was too ill to continue. The adit was therefore possibly fouled with enteric evacuations for sixteen days, and possibly in increasing amounts. The epidemic began on January 19th, and reaching its climax on February 1st came to an end about a month later.

Although I think that Sir Richard Thorne clearly demonstrated the true source of the enteric contamination, it was nevertheless shown that the Caterham wells (three in number, sunk for some 400 or 500 feet through clayey

gravel and chalk, and with borings going to a greater depth) were seriously liable to contamination through fissures. Thus on one occasion "waste water was pumped into a hollow spot about 170 feet to the east of the wells; here it disappeared, but it was soon ascertained to be returning into the wells at a depth of 420 feet." The formation in which the wells were sunk was, indeed, dangerously fissured in all directions, and there were cesspools at various spots round the works.

3. At *Beverley* the well (333 feet deep) was alongside a sewage farm belonging to the East Riding Asylum, and from an irrigated field sewage is supposed to have trickled down the well shaft. Here we have an instance of sanitary works carried out for the benefit of one community causing serious trouble to their neighbours; and other instances of this have occurred.

4. At *Hitchin* we are told that defects in the water-works permitted occasional back-flow of sewage-polluted river water into the pumping well.

5. At *Margate* the well was exposed to contamination by soakage from sea water and from cesspools.

6. At *Worthing* we have a leakage of a sewer into a heading run from the bottom of a deep chalk well.

7. At *Newport* (Isle of Wight) the chalk wells were fouled by an adjacent pond, and probably by more distant leaking cesspools, of which there were many. This water contained the *Bacillus coli*, and I trust we may learn in this discussion what is the true value of that fact.

The eighth case of the fouling of a deep well occurred at *Herrington*, near *Houghton-le-Spring*, and the following are the facts:

1889. *Houghton-le-Spring* (Dr. David Page).—258 cases occurring in a population of 4400 situated within the area of the *Herrington* water-supply.

The supply was from a well 330 feet deep, through

"clay with sand partings in seams" (72 feet) ; limestone marl (15 feet) ; and for the rest through sandstone and shale. The sides were lined with bricks set in mortar, and it was believed to be impervious.

On examination the sides of the well were defective, and a "feeder" was found discharging into the well 45 feet below the surface.

Microscopic examination of this water afforded evidence of contamination with household refuse, and the source of contamination proved to be a farmhouse containing a water-closet, situated three quarters of a mile off and draining into the well through a fissure in the magnesian limestone caused by the subsidence of a colliery beneath.

On examination the pipes of the Herrington service were found excellently jointed, but the sewer pipes were leaking.

This case is one of very great importance. The water company here was free from blame ; the pollution was due to what may be called a pure accident.

It is well to call attention to this case, because the deep tunnelling which is going on, and which is being proposed in London, may not be without its effects upon the network of pipes above.

These facts lead one to make a few remarks as to sources of water. Enteric fever being essentially a human disease, the chances of getting water impregnated with enteric at its source must bear direct ratio to the density of population round that source. On that account there is just now a rush for gathering grounds which are uninhabited. Some of these moorland waters are not without their disadvantages, and we know very little as to the comparative receptivity for poison in hard or soft waters in the presence of enteric excreta. In the above list there are three instances (Swansea [2] and Paisley) of contamination of upland waters.

I find in Glasgow, which enjoys an unlimited supply of the purest water in the world, that in the ten years

1881-90 the enteric fever death-rate was  $\cdot 25$  per 1000 living. The enteric death-rate for 1881-90 for England and Wales was  $\cdot 20$ , and for London  $\cdot 19$ . The figures seem to show that Glasgow, in spite of its pure water, enjoys no immunity from enteric fever.

With regard to the relative advantages of wells and springs (captured and enclosed) as against rivers, there are certain obvious facts which must not be forgotten. If a well or spring be fouled, the whole of the fouling is mixed with the water and supplied to the houses. There is, so to say, no escape for accidental pollution except into the water-pipes. Given the fact of fouling, wells and springs are likely to deliver a dangerous dose. A well, in this respect, is analogous to the "dead end" of a water main, which is always a dangerous spot in case of fouling. If a river be fouled, more or less floats away or passes by the intake. I need hardly allude to the obvious fact that the water in a river is exposed to fresh air and sunlight, while the water in a well enjoys no such advantage.

#### POLLUTION IN THE COURSE.

There were 18 cases in which a public water-supply was polluted in its *course*, that is, in its transit between the source and the houses, viz. at Guildford, Tideswell, Dewsbury, Haverfordwest, Ventnor, Blackburn, Bangor, Sheerness, Kidderminster, Mountain Ash, Lewes, Hull, Over Darwen, Perth, Fareham, Buckingham, Atherstone, and Worthing. Of these 18 cases, 1 (Bangor) was caused by fouling of the filter-beds, and 2 (Haverfordwest and Ventnor) were due to fouling of the reservoirs. The other 15 were practically due to the same cause, viz. *the in-suction of foul air or liquids by leaking water-pipes during intermissions in the supply*. It is not necessary to go into details of these cases, but it may be stated that at Guildford a sewer was actually traversed by a water-pipe, and at Sheerness "water mains were often

laid in the same trench as tributary sewers." At Perth river water from near the sewer outflow got into the water main which runs under the Tay.

We must remember that sewer-pipes and water-pipes, however well laid and made, are like ourselves mortal, and liable to accidents and the inevitable results of age. They are perfectly sure to leak sooner or later from some cause or another. If they leak we must reckon upon an interchange of their contents. These pipes are always placed in proximity to each other and *out of sight*, so that if the sewers leak into the water-pipes our first indication of it is an outbreak of disease.

I have a cottage in the Thames valley where the Local Council, by the help of some £130,000 borrowed with the consent and implied approval of the Local Government Board, is and has been busy by dint of sewerage and paving all the country roads, in making the way smooth for the speculative builder and site owner, who fill their pockets by building houses with the minimum curtilage allowed by the Local Government Board. The houses are springing up at a rate of twenty-five or thirty to the acre, and at a rent of about eight shillings a week. I see the water-pipes and sewer-pipes being laid for this mushroom crop of brick and stucco, and in the mornings I hear the traction engine and steam roller rumbling about only a few feet above these pipes, as though their only thought was to make quite sure of cracking them.

I may recall the fact that a few years ago in London the local authorities, by removing every particle of snow and throwing down salt, succeeded in surrounding our water-pipes with a freezing mixture, with the result that no small proportion of them burst; and I may also recall the fact that a deep tunnel caused a dangerous subsidence of the earth above it only a few weeks ago in the City. The only way of minimising the very obvious danger of thus laying water-pipes and sewer-pipes would be to insist that where they go together they shall do so only in subways, where they are open at all times to ready

inspection. An alternative would be to insist on maintaining narrow roadways between the backs of new houses, and to keep the sewers at the back and the water-pipes in the front. Whatever plan be adopted, it is evident that these two sets of pipes—afferent and efferent—must come into close proximity somewhere. Nevertheless what I have suggested would lessen this danger.

#### POLLUTION AT THE PERIPHERY.

Next I have to say a few words on the very important subject of the pollution of water at the *periphery*, *i. e. through the taps*.

This mode of pollution has been, I think, most conclusively established.

One of the first persons to call attention to this danger was Dr. Blaxall, in a report on an outbreak of enteric fever at Sherborne. This was followed in a few months by the late Sir George Buchanan's report on Caius College, which must remain for all time one of the classics in the literature of preventive medicine. Mr. Spear in his report on Mountain Ash, Glamorgan, deals with the same point. I will quote from these three reports, but I think we shall derive most instruction by reversing the order and taking Mr. Spear's first.

1888. *Mountain Ash* (Mr. John Spear).—The 518 cases of this epidemic were chiefly due to the in-suction of foul air (and liquids and solids?) into water-pipes during intermissions of supply. Leakages in the pipes were numerous.

There was some evidence to show that streets supplied with water-pipes having "dead ends" suffered somewhat more than others. Concerning one of these terminal "dead end" branches Mr. Spear says, "It would take its full share of the first rush of water (after an intermission), and all impurities would have to be withdrawn through the house taps."

In another place he says, "Given a fever poison present in a water system, it is quite possible that in the dead ends of pipes or at other places where stagnation occurs, or at any given point where escape may take place into the soil, further development of the poison leading to new or more intense infection of the water may occur."

Allusion is made to a dead-end pipe, fifty-three yards long, gradually ascending from a larger main to supply a school, including two closets which were supplied by pipes *direct from the main*. "When the water was turned off at the main, and the tap of one of these closets opened for a moment, suction of air up the pipe was immediately heard." Mr. Spear further adds in a foot-note, "suction into this main was very strong during intermission of service. At such times the house taps greedily *sucked up water* placed at their open mouths." It wants only a moment's consideration to see that ascending "dead ends," whether in houses or in streets, are converted into powerful aspirators directly pressure within them fails.

In the school above mentioned there were two cases of enteric, and it is possible that specifically infected matter had been aspirated into the mains.

In another part of his report Mr. Spear has this important paragraph:—"Intermission of water-current, however, is not by any means essential to the introduction of foreign matters into water-pipes. Under various physical conditions very powerful in-suction of external matters into a full-flowing water-pipe can take place. . . . But the fact of this lateral in-suction into water-pipes 'running full' is not universally known as it ought to be to many of the engineers who undertake the responsible business of laying water mains and other water-pipes."

1873. *Caius College* (Dr. Buchanan).—15 cases, of which 12 occurred in "Tree Court," which had been built only a few years and contained all the latest



"sanitary improvements." Out of 63 residents in Tree Court, 12 (or 19 per cent.) were stricken with fever in the month of November. Tree Court had a water-supply through a branch from the main in the street, which, to use a pathological expression, was an "end artery," and although it supplied a branch to each of the nine staircases of Tree Court, formed no anastomoses with any other water-pipes in the college. This water got fouled by one of two private closets in Tree Court. The manner of the fouling was in this way. The closet which was on the first floor of staircase P was provided with a "safe," *i. e.* a big metal tray surrounding the base of the closet to catch accidental splashings and drippings. This "safe" drained into the soil-pipe just beyond the closet-trap, through a small pipe with a trap of its own. The subsidiary trap was fed by what is technically known as a weeping-pipe, a branch of the pipe supplying the closet. This closet supply was direct from the Tree Court main without the intervention of any cistern.

The water-supply to Caius College is "continuous," but it is liable to intermissions, and such intermissions are known to have occurred on October 25th and November 1st, and probably at other times.

When, with the water turned off, the closet on staircase P was used, the trap of the safe would be emptied and the trap of the closet be left filled more or less with fæces. The weeping-pipe was therefore open to the sewer air coming up the soil-pipe and to the filth in the closet trap. With the pipes of Tree Court empty of water they would be liable to be filled with sewer air. It was further proved that the end of the weeping-pipe was covered with filth, and it is probable that the water system of Tree Court was definitely inoculated with *Bacillus typhosus* in addition to being impregnated with sewer air.

1873. *Sherborne* (Dr. Blaxall).—243 cases. "Board of Health" water derived from two deep wells. Water-

closets supplied direct from the mains. Between December, 1872, and May, 1873, there were 243 cases. "During December, 1872, and January, 1873, the water was frequently shut off from the town at a point near to the reservoir, and the same thing was done every night in February. It is known that when the water was thus shut off there were certain delivery pipes up which there was a rush of air immediately the tap was unscrewed. Now many of the openings of the pipes, as before described, are situated in the pans of the water-closets. At night, after the closet had been used, the tap would be turned on for the admission of water; none flowing, the tap in many instances would not be turned off again, and thus a direct passage into the water mains would be left open. . . . Thus the system of pipes for the water-supply became the means of ventilating the closet pans; if a trap happened to be broken or out of order it became a means of ventilating the sewers, and if a pan happened to be full of excrement that excrement would be sucked into the water-pipe."

This mode of water pollution is well understood, and we know the importance of maintaining constant pressure in our water service. It seems clear that many of the critics of Maidstone have not been aware of the frightful danger of lessening water pressure at a time when the sewer-pipes must have been highly charged with enteric poison.

How far is the maintenance of constant pressure attainable? It is evident that there *must* arise occasions when the water-pipes *must* be empty. To maintain constant pressure is a counsel of perfection which is unattainable.

The dangers of peripheral pollution, by the in-suction or diffusion of foul gas or liquid, is so well recognised that all w.c.'s are now supplied through independent cisterns.

The recent unfortunate outbreak at University College

Hospital may be mentioned in this connection. The facts were briefly these:—Twenty-one nurses and nurses' servants were in the first week of October attacked with enteric. In consequence of the rebuilding of the hospital, now in progress, it had become necessary to fit up a temporary dining-room in the basement for the nurses. A sink was wanted, and the water for this sink was taken by a branch from the nearest water-pipe. These alterations were made in September, and no advice was sought concerning what appeared a trivial matter from anybody having a knowledge of sanitation. The water-pipe was a descending pipe coming from a cistern standing in the closet lobby of a surgical ward on the floor above. This pipe had three branches: (a) about four feet below the cistern for the supply of a slop sink; (b) about twenty feet below the cistern for the supply of the temporary sink above mentioned; and (c) the terminal tap of this system which supplied the tank in which the enteric linen is placed in carbolised water at a distance of fifty feet from the cistern. In order to insert the branch (b) into the pipe descending from the cistern all the water was run off, and the tap over the tank was left open *while the cistern was empty*. It is not possible to believe that there could have been any aspiration of gas or fluid under this arrangement, but air may have travelled up this fifty feet of pipe into the cistern *while the latter was empty*.

While the cistern was empty the plumber got into it in his ordinary clothes and boots and cleaned it thoroughly, and of course loosened whatever films there may have been on the bottom and sides of this cistern. Then the taps were closed and the cistern was filled, and the evidence goes to show that those who drank from this cistern immediately after its "disturbance" by the plumber in his ordinary clothes and boots were smitten with enteric. Now the impregnation of this empty cistern by air travelling along fifty feet of pipe must have been very trifling, and such air must have been dis-

placed by the intrushing water when the cistern was filled. But who would like to drink the first water from a cistern located inside a hospital just after it had been stirred up by a plumber in his ordinary clothes and boots? The water from the cistern was carefully examined by Dr. Curtis, and no trace of *Bacillus coli* or any putrefactive organism was found. The water examined was taken some three weeks after the occurrence which I have detailed. If Dr. Curtis could have got the water of September 13th, the result would in all likelihood have been different.

Although I think that the cleaning of the cistern had more to do with this particular outbreak than the passage of air up the pipe, I am well aware that the passage of air (without a previous vacuum) up pipes, or rather the passage of enteric poison up pipes (possibly by the growth upwards of a film), is a fact which may almost be regarded as proved.

There is yet another way in which peripheral pollution of a public water-supply may take place.

The outbreak at Worthing in 1893 was accompanied by an outbreak in the adjoining districts of West Tarring and West Worthing having a different water-supply, and the explanation of this is that Worthing water entered the pipes of the adjoining districts through the ball hydrants in the street at a time when the pressure in the pipes intermitted.

A few weeks before the close of the last holidays I was staying at Andover, which has a municipal water-supply obtained from a deep chalk well. During one of the last days of September there was a very heavy thunderstorm with torrents of rain. I went out in this storm to one of the lowest points in the town, and there I found what I had found before under similar circumstances, viz. the water bursting up through the street sewer, with the result that the street was flooded with foul water and faecal matter. A few inches below this mess were the water-pipes supplying the district, and I

hope they were not cracked, and that the pressure in them was fully maintained until after this impregnating flood had subsided.

I stood at the lowest point to which storm water must gravitate, and here nature had provided an overflow into a ditch. Some years ago the storm water was taken into the sewer, and a storm overflow from the sewer was taken into the ditch. Nature meant that the sudden storm should cleanse the surface of the ground and also cleanse the ditch, and thus do nothing but good. Man interfered, with the result that instead of being cleansed, both the street and the ditch were filled with the poisonous purgings of a sewer. I give this as a type of what is common. The craze for taking rain water underground instead of allowing it to run on the surface is as dangerous as it is costly.

The experiences at Sherborne, Mountain Ash, and Caius College seem to point to the conclusion that every public water-supply should *protect its own purity*, by compelling every house to have a cistern of its own as a "cut-off" between the impurities of the house and the water in the mains. This necessity is implied by the compulsion which we are now under of having a separate cistern for the water-closets. But water-closets are not the only filthy places. There are water-taps which are theoretically capable of tainting the water-pipes under certain given conditions almost everywhere. Most "noxious trades" require water. Kitchen sinks are not always sweet; the post-mortem room sink is an uncomfortable thing to think of in connection with peripheral pollution, and bacteriological laboratories fill us with "horrible imaginings." Sewer air is everywhere over street gratings, and at the eaves, and although diluted almost to vanishing point, it makes up by quantity what it may lack in intensity.

In Dr. Thresh's valuable book on 'Water-supplies,' p. 211, I find an allusion to an observation by Sir G. Buchanan at Croydon in 1875, of an instance in which

bloody water was drawn from a tap at a house next door to a slaughterhouse. This is a fact which is very eloquent in connection with automatic peripheral pollution.

Under existing conditions it appears certain that every common water-supply is to a greater or less extent automatically self-polluting, and it will be interesting to hear what the sanitary officials may have to say on this question of the house-cistern *as a protection to the general supply*.

A dirty house-cistern is doubtless very undesirable for the people in the house. If it is dirty, that is their fault, and it seems unreasonable that the purity of the general supply should be endangered by the filthiness or carelessness of the householder.

That the filthy should "stew in their own gravy" is reasonable enough, but that the cleanly should be called upon to stew in it is unreasonable.

#### RECOMMENDATIONS AS TO WATER-SUPPLY—WHAT TO DO WITH INFECTED MATERIAL.

The remarks which I have made on public water-supplies seem to show that when we accept a water-supply from a public body, our health to a considerable extent passes out of our own keeping. That is obvious; and it is also obvious that when the order is given to close a private well, and to accept a public supply, the sanitary authority incurs a very grave responsibility indeed.

The instances I have quoted seem to compel the conclusion that public water-supplies are a cause of the epidemicity of enteric fever. These water-supplies diffuse the poison with a completeness which is almost inconceivable, and further insure that the poison is laid on, as it were, to our very mouths.

The condition of a city with its water-supply polluted

by enteric is fearful to contemplate. Not only is the water being drunk, but the inhabitants are washing in it, the dairies and milk cans are being swilled with it, the very pats of "margarine" are being made up with its help, the oysters are lying in it, the salads and flowers are being sprinkled with it, and the streets are being watered with it. Then when the need of closet-flushing is at its maximum, and the street sewers are full of enteric poison, comes the order to cut off the water, and it certainly requires great skill to minimise the danger of so doing.

These epidemics show the danger of "putting too many eggs in one basket," and they force upon us the necessity of arranging water-supplies with as many independent subdivisions as may be feasible. They also serve to show the necessity of not permitting the mixing of water derived from different sources. Dairy companies have learnt, by bitter experience, the necessity of keeping the milk-supply from different farms absolutely distinct. Water companies must do the same thing.

The great milk companies have voluntarily adopted a system of milk supervision which approaches perfection. The milk is submitted to a daily, almost hourly examination by skilled experts.

The water companies must do the same thing. Every water company ought to have a laboratory for the chemical and bacteriological examination of the water which it distributes, and there ought to be a proper staff of skilled experts *constantly* at work examining samples taken daily, or even hourly, not only at the source, but at various points in the area of distribution.

Under existing conditions the purity of our water-supplies can only be maintained by vigilance and publicity. The notification of disease is undoubtedly a great protection, but it is clear that *every notification should be forwarded instantly to the water authority and the sewer authority, whose respective inspectors should examine the implicated premises without delay.*

It is evident that repeated chemical and bacteriological examinations of water are of great value, but it is not probable that they are much protection against sudden pollutions occurring after heavy downpours of rain. The mischief is done suddenly, and both the chemist and bacteriologist will possibly be able to register the catastrophe, but not prevent it. It is most important that the public should not be led to expect the impossible, and to imagine that analyses can make a public water-supply secure. The interpretation of analyses is not always easy, but any sudden change in the quality of a water would arouse suspicions.

Of what import is the presence of *Bacillus coli* in a water? This bacillus is known to occur in rabbits and birds. Does it occur in the dung or dead bodies of insects, such as spiders, centipedes, slugs, snails, &c., which are very apt to gain an entrance into wells? Does it occur in fish? This question ought to be answered.

All sources of water not only need to be guarded by chemists and bacteriologists, but they need constant watching by a shrewd practical man. It is not too much to say that all sources of water have their purity endangered by sudden flood and downpours, and water-collection should be as far as possible stopped during such occurrences. A clear water which suddenly becomes turbid must always be dangerous. My shallow well at Andover, which is only five feet deep, and which is absolutely protected from surface or lateral contamination, has once or twice become turbid after exceptionally heavy rains. The reason for this appears to be that so soon as the soil above the ground water gets saturated, the deposit on the bottom of the well is driven upwards by the suddenly increased pressure under which the water rises in the well.

There is very definite safety in having the water sources near at hand and capable of being inspected. By means of the electric light a well of moderate depth



can easily be inspected, but it is difficult to know what may have happened in a bore-hole at a depth of 400 or 500 feet.

There are some who are of opinion that the public safety would be enhanced by placing our water-supplies in the hands of public authorities ; I am not of this opinion for the following reasons :

1. Because private authorities can control their servants and get rid of incompetent officers more easily and promptly than public bodies, who are elected to their offices by their servants and the friends of their servants.

2. Because water companies equally with railway companies should be made liable to "damages" in case of accidents. A successful action for damages against a sanitary authority by, let us say, 10 per cent. of the ratepayers of a town would result in the payment by the plaintiffs of 10 per cent. of the damages awarded them, while the bulk of the remainder would be paid by persons who are blameless and helpless. This is a *reductio ad absurdum*.

3. Because no person should be allowed to control a water company without incurring some degree of personal responsibility. It is the power of borrowing and spending money without personal responsibility that constitutes the dangerous element in our modern local government.

I am very strongly of opinion that water companies should be placed exactly on the same footing as other trading companies, and that every householder should have the right of paying for water by meter if he so desires. In houses not supplied by meter no waste-pipe should be allowed beneath any supply tap, so as to insure that, if the tap be left carelessly turned on, the house must be flooded. This would effectually check waste.

It seems very necessary to check waste of water. Many local authorities are at their wits' end for water, and are calling in the help of wizards. But we have had the comforting assurance that this, at least, is a form of

sanitary quackery which the public auditors do not allow to be paid for out of the rates.

Wilful or careless waste of water (as by leaving a tap turned on) should be punished by fine when water is not paid for by meter.

Waste of water often necessitates the severe pumping of wells and other sources, which increases the danger of pollution. Waste must be checked.

There are others who seem to think that we shall attain greater safety by the registration of plumbers. These sanitary scapegoats are, I think, more sinned against than sinning, and are often blamed most undeservedly for the inherent shortcomings of our modern sanitary methods. If, however, plumbers are to be registered, it does seem desirable that they should be divided into two classes, and that we should run no risk of allowing a man to attend to the water service who perhaps carries about him infective matter accidentally picked up in the "drains."

#### THE TREATMENT OF INFECTED LINEN.

At University College Hospital some years ago this question was submitted to a small committee, of which I was a member, and we advised as follows:

1. That the linen should not be treated with carbolic acid or corrosive sublimate, because of the fear of coagulating albuminous soilings, and for the same reason that it should not be put at once into boiling water.

2. We advised that a special tank should be provided, and should be filled every day with cold water to which washing soda is added (about two pounds to a hundred gallons); into this tank the soiled linen is taken directly from the wards, and is allowed to soak till the following morning, when the water in the tank is thoroughly boiled for one and a half hours, and after boiling is run off into the sewer. In this way the first stage of the laundry

work is accomplished. The effluent water is sterilised, as is also the linen.

The same process can easily be carried out in private on a smaller scale.

#### DISPOSAL OF EXCRETA.

We now come to the important question as to what ought to be done with the stools of enteric patients. I have no hesitation in saying that in towns at least they should be *burnt*, and in all fever hospitals intended for the reception of this class of patients a proper destructor should be provided.

I have very little doubt that if this be accepted as a principle, human ingenuity will soon provide the means.

It is most desirable that, in the interest of the nurses and friends, there should be a minimum of manipulation of enteric fæces. I have my own ideas as to how this end may be accomplished, but I must limit myself to the discussion of principles rather than details. I have very little faith in chemical disinfections. Badly done this is more likely to preserve than destroy the poison, and the amount of manipulation which thorough chemical disinfection necessitates is most undesirable.

Of one thing I am convinced, and it is this, that *under no circumstances whatever should enteric excreta be mixed with water*. We know how insidious this mixture is, and that the danger of it practically increases when dilution has been carried to the extent that the mixture has, to our unaided senses, the characters of potable water. Enteric fever is pre-eminently a water-borne disease. Enteric stools, therefore, must not be mixed with water, because it is the property of water to return to its source.

“ All things that are of the earth turn to the earth again,  
And all things that are of the waters return into the sea.”

*Ecclus. xl, 2.*

Let me illustrate this by some examples. A stool mixed with water flows to the sewer, and so directly to the sea. One would suppose that a stool so treated was safely disposed of. Not at all; the enteric poison returns to us upon the oyster, and it must be admitted that our habit of mixing enteric stools with water is in a fair way to destroy our oyster industries—the fame of which extends to the extreme limits of our historic record. A very eminent agriculturist has said that our sewage is good for our fisheries. At present we have very conclusive evidence of evil, and no evidence of good of any kind.

Again, an enteric stool mixed with water flows to the sewer, and from the sewer leaks into a deep chalk well, as at Guildford and Worthing, or runs off the surface of a sewage farm into a deep well, as at Beverley, whence it is pumped in a state of extreme subdivision to poison others. Or the mixture runs to the sewer, and so to the river, coursing *en route* through the watercress beds. From the river it is pumped to the consumer of water, and the watercress is sent far and wide, mainly to our operatives in the big towns. The watercress industry, like the oyster industry, is endangered, and we may soon be deprived of this most agreeable vegetable. Or the mixture runs to the sewer or cesspool, and leaking, *under pressure*, worms its way sooner or later to a well or spring, and possibly finds its way to the milk. Or the water leaking from the sewer flows into the leaking water-pipe, and so back to the consumer. Lastly, as at Caius College, the mixture may be sucked directly from the water-closet into the pipe supplying the drinking-water.

Water returns to its source, whether the circuit be long or short.

If any underground receptacle for water or sewage begins to leak, and if the water in such receptacle be constantly renewed, there is no limit to the distance whether laterally or vertically which it may not travel. The danger of a constant drip of water on one spot is proverbial.

It is a very serious danger in relation to our water-supply. The deeper it goes, the further it is likely to go. The pressure of a column of water is, roughly speaking, nearly equal to half a pound for each foot of vertical height. The force with which the water returned from the surface into the Caterham well was, at its maximum, about 185 lbs. to the square inch.

What has just been said is founded on facts which are familiar to everybody, and which are of almost daily occurrence, and in this discussion it is most essential to keep fact and theory quite distinct. The familiar accidents to which I have just alluded are due entirely to the fact that enteric stools have been mixed with water.

#### HISTORICAL.

There can be no doubt but that enteric fever must have existed from all time, and that stools must often have accidentally found their way to a spring or other water source, but I think there is small room to doubt that the great cause of the increase and wide epidemicity of enteric fever in modern times has been the water-closet.

It is now nearly half a century ago (in 1849) that my great master, Sir William Jenner, contributed to this Society a paper which closed the controversy which had been raging for some ten years previously on the identity or non-identity of typhus and typhoid fevers. The distinction between them is so clear that we of to-day can hardly understand the controversy. It has been like a dissolving view; the clearly focussed typhus of 1830 became obscured by new elements, which have gradually entirely occupied the screen, till now we have nothing but the distinct features of enteric, and typhus has vanished.

It seems probable that the main cause of the vanishing of typhus was the repeal of the Corn Laws in 1846, and the better wages and cheaper food which our industrial population has enjoyed in increasing degree.

On the other hand, the rise of enteric was probably due to the invention of the water-closet and the gradual introduction of water-carried sewage. It must be remembered that the year 1849, in which the non-identity of typhus and enteric was settled, was the year in which cholera was raging, the year in which the great fact of water-borne disease was established.

The conditions which gave us the great outburst of cholera in 1849, when the Thames between the bridges was at once our cesspool and our source of water, were eminently calculated to furnish sufficient enteric fever for the establishment of its identity. If the same conditions had obtained in the days of Heberden or Mead, Sydenham or Morton, the identity of these fevers would have been established one or two centuries before. So glaring a fact could not have escaped these most acute observers.

The sanitary methods of our ancestors were simpler than ours. Water was pumped from a well on the premises, excrement was deposited in a *dry* privy and removed once a year by the nightman, while the slops—trivial in amount as compared with the present day, and containing no specific poisons—ran to a dead well or sump, or flowed in an open channel to the nearest stream. The old-fashioned privy, *if kept scrupulously dry* and if the vault was ventilated, was by no means so offensive as is the cesspool. The smell was *sui generis*. It was generally well removed from the house; sometimes was big enough to accommodate three people at once; was furnished with a special seat for children (a most excellent arrangement), and sometimes had a courtyard of its own. There was no sloppiness or putrefaction, and not much or any soakage, especially if the vault was ventilated so as to allow of evaporation. When this arrangement was carried out with a fair amount of common sense and with sufficient space the surface-well ran little risk of infection by specific poisons.

Water being unobtainable without the labour of pumping, there was no waste, and allowance being made for

evaporation, *the volume of slop-water must always have been less than the volume pumped from the subsoil.* This appears to me to be a not unimportant fact, and it is evident that the soil round the dwelling ran less risk of getting water-logged and sour than is the case where unlimited water is poured into the premises of thriftless and careless people, who are incessantly pouring slops into a subsoil from which they are forbidden to pump.

When the water-closet was introduced, and its contents were shot into the previously dry privy, fearful putrefaction resulted. "Sewer gas" was invented, and the privy began to leak under pressure, to the imminent danger of the well. The privy was, by the use of the water-closet, converted into a cesspool.

As late (I quote Mr. Lewis Angell) as 1815 it was penal to discharge house drainage into sewers. Chief Justice Holt laid it down "that every man should keep his dirt to himself."

The water-closets and the resulting cesspools necessitated first the admission of the *overflow* from cesspools into the sewers, and then about 1848 some 30,000 cesspools were abolished in London, and the houses drained directly into the sewers and so to the Thames. We have had cholera in 1832, 1849, 1854, and 1866; the death-rate from diarrhœa, which in London was .215 in 1838, rose to 1.705 in 1849.

As a direct consequence of the legislation of 1848, every river and every source of water in the country became polluted or in danger of pollution. We then passed a law forbidding the pollution of rivers.

Dr. William Farr fully recognised that the cholera of 1849 and 1854, and the increase of diarrhœa, were due to the water-closet, but he still hoped that the contents of a machine which rids our houses of filth need not necessarily poison our rivers and other sources of water.

I confess I am very pessimistic on this head, as will be gathered from my remarks on the property of water to return to its source; and I feel convinced that unless we

are guided by sound principles, all expenditure with a view to the prevention of enteric fever will be wasted.

In the account of almost every outburst of enteric fever one comes to the inevitable mixture (generally deliberate and intentional, but sometimes accidental) of fæces and water, and in a large majority of such cases we find the water-closet. There are some who seem to think that the abolition of middens and cesspools and their replacement by the underground sewer will abolish enteric. I cannot share any such belief, because it has been shown again and again that the leaking sewer or drain is quite as much a danger as the leaking cesspool, and water leaking from the same spot may (as I have shown) trickle any distance. A drain made of so-called sanitary pipes has a potentiality for leaking at some 1760 joints in every mile of length.

Every town or village which has underground sewers will certainly, sooner or later, poison its local wells and other sources of water.

Whether or no we are able to obtain pure water depends entirely upon our treatment of putrescible refuse. If this be put beneath the surface of the ground instead of upon it, the danger to every source of water is immense.

#### APPLICATION OF STOOLS TO WELL-TILLED HUMUS.

In country places I believe that enteric stools may with reasonable safety be applied to the surface of well-tilled soil. They must not be mixed with antiseptics, and they must on no account be buried deeply. They must be placed only a few inches below the surface, and be lightly covered. *There is no evidence, so far as I am aware, that fæces (enteric or otherwise) treated in this way have ever been productive of harm.* The sooner the ground is planted with cabbage plants the better, and it must be borne in mind that, if the soil is to remain sweet, tillage and cropping are a *sine quâ non*, and it is probable that



pathogenic microbes succumb in the process. There is no evidence to the contrary. Tillage admits air, and in turning over the ground we have the beneficent effect of sunlight. I have studied somewhat closely for the past fourteen years the result of applying human fæces to a well-tilled humus, and I feel convinced that many who write upon the subject have taken but little pains to inform themselves as to the real facts. The rapidity with which fæcal matter disappears in the earth depends upon circumstances, viz. the state of the soil as to tillage, the mass of fæces to be dealt with, and the weather. If the weather be warm with occasional showers, we find at the end of about three weeks that the whole of the fæces and paper has become humified, and is no longer recognisable by eye or nose. The smell under all circumstances seems to disappear in a few hours. If there be drought or frost the humification is delayed, but, strangely enough, the humification is delayed longest if the weather be continuously wet. Under these circumstances the pores of the soil are closed, and the humus gets sticky and pasty, but the fæces are not and cannot be washed out of the soil. On the top of the soil, water can exert no continuous pressure. What harm are fæces capable of doing when thus applied to the soil? They are not likely to be eaten or inhaled, they cannot be drunk, and nothing short of absolute proof would make me believe that the bacilli can pass downward to the subsoil water, because, in well-tilled soil, the harder it rains the tighter are the fæces locked up, and with them one must suppose the bacilli, so long as they continue to exist. I have never studied microscopically what one may call the progressive fungology of fæces on their road to humification, but I can say this, that on the surface of the ground in the course of a month or so there appears a green algoid growth, looking somewhat like a covering of moss, and then on digging and taking up a handful of earth one may detect little particles riddled with fungi, often reddish and ultimately black. The end is what the

gardener calls "good rich black mould." Its fertility is unequalled. I must repeat what I have said, that the place of deposit must be *well-tilled humus*. If masses of fæces are dumped down on clay banks sloping to a river they may be washed into the river, and have been washed in many a time. Or they may be driven by torrential rain into deep cracks in the clay, and this has possibly happened in India and perhaps here also. Again I feel tempted to put forward a hypothetical case. It is this: as long as plants are alive their roots absorb moisture from the soil, but when they die the dead root of a tree or strong-rooted plant like the hop may possibly serve as a drain for the direct conduction of moisture to the subsoil. This is purely hypothetical, and I have no knowledge of any such occurrence. In a properly tilled humus there are no cracks or fissures, and any cracks must inevitably be filled up by the crumbly earth. The power of a well-tilled humus to absorb, transform, and humify organic matter is astounding; and, provided it be tilled and cropped, its power in this way seems to steadily increase, as if "increase of appetite did grow by what it fed on."

In my published works ('Essays on Rural Hygiene' and 'The Dwelling-house') will be found details of experiments which show conclusively how great is the purifying power of humus on urine which is slowly filtered through it; and, in short, there is abundant evidence that a well-tilled and well-nourished humus is an absolute protection to our subterranean water-supplies. Further, I believe that, so long as the humus keeps healthy and in a highly productive state, the protection to our water-supplies is proportionate to the thickness and richness of the humus, or, in other words, to the amount of dung applied to it.

Further, I believe, and have supported the statement by arguments in the works referred to, that the best return agriculturally is got by the immediate and daily application of fæces to the land, and that while the fæcal

matter is, in agricultural phrase, 'ripening,' one may get a crop of cabbages, and after the cabbages anything and everything. These statements are founded on an experiment now in its fourteenth year, in which the fæces of about one hundred people have been utilised in the manner indicated on an acre and a quarter of land. Now that the garden is in full bearing it returns me about fifty pounds a year.

Is it not time that the people of this country were taught that fæces properly used are a source of health, wealth, and beauty? This most important matter holds no place in our National Educational System. There are no municipal gardens for showing how fæces may increase the attractiveness and prosperity of a town, and to demonstrate that the man who resolutely applies fæcal matter to its proper use gets an immediate and great reward. The people are silently taught that the only "decent" (!) way of dealing with fæces is to mix them with water. This filthy mixture, by its putrefaction, fills our courts and alleys with sewer gas; it trickles to our water sources, and the expensive works necessary for attempting to sweeten it and purify it are of such a horrible kind that they are always surrounded by walls which reach higher than the nose and eyes of an ordinary man. I have always contended that there is too much of Hercules and too little of Minerva in our sanitary arrangements. We see the pipes, the engines, the ventilators, the hospitals, and the smoke of the destructor; we hear the incessant thud of steam machinery, and feel the rate collector's hand for ever in our pockets, but we never get a glimpse of the bright side of the matter, the return which nature inevitably makes to nourish our bodies, gladden our senses, and freshen the air. Alas! the municipal engineer is too often a person who abhors nature and loves a vacuum.

## POLLUTED SOIL.

In the reports of the Local Government Board Inspectors we constantly come across allusions to "a polluted soil." The water-closet has polluted our rivers and the air we breathe; we now begin to hear of polluted soil. The outlook is not pleasant. Sir Richard Thorne, in a recent most interesting address, alludes to experiments carried out by my friend and colleague Dr. Sidney Martin, which show that the typhoid bacillus will thrive and multiply in a rich soil *previously sterilised*. This is an interesting fact, and I find no difficulty in believing that in the untilled soils, sodden with fæcal and other filth, which are found in the close courts of our modern manufacturing towns and in the old quarters of towns which in former ages were walled fortresses, the enteric poison may lurk. There is no more difficulty in believing that the enteric poison will live in sterilised earth than in believing that it will live in gelatine, which has proved a valuable cultivating ground for so many microbes. Acting upon this knowledge, Dr. Ballard showed that cold gelatinous food stored in an unwholesome place might become a cultivating ground for pathogenic organisms, and he published many facts in support of his contention. But it would be quite as unreasonable to condemn jelly as necessarily a dangerous food as it would be to condemn the soil as the natural lurking-place of enteric because under conditions which cannot exist in nature the enteric microbe may exist in it.

It is necessary to state this because a careless reader of some of the Local Government Board Reports might come to the conclusion that the manuring of the land was a serious danger to the public health. A moment's reflection will convince us that the balance of effects resulting from the dunging of land must be in favour of health, and there is no evidence of evil resulting from farming or gardening operations. Agriculturists of all

grades are the healthiest section of our population. The great cities would be in a most pitiable state if the gardener were not ready to cart away the dung and bring back fresh fruit and vegetables. As the dung of the modern horse bids fair to be cinders, one hopes that a demand will spring up for human fæces, and that the necessary machinery for their decent daily removal will be accomplished.

#### TOO MUCH WATER.

The soils of cities become polluted because there is a deficiency of air and sunlight and an excess of water. The careless inhabitants can get water by turning a tap. They throw it down the middens, they cast it on the ash heaps, they spill it on the surface. The subsoil is never relieved of water by pumping, and foul liquids are sure to be soaking into it from cracked drain-pipes. Nothing can purify a filth-sodden soil except tillage, and I much doubt whether an impermeable paving will do any good if there be a chance of drain-pipes leaking beneath it. In some of our filthy industrial sties no sanitary measures short of destruction can possibly do much good, and I fail to see the use of herculean and endless labour in order to secure the survival of the unfittest, unless we give the possibility of wholesome life, which cannot be attained without adequate space.

In these places the soil has become polluted because fæcal matter has been allowed to escape the action of the humus, and being mixed with water has been deliberately taken through to the subsoil, out of the reach of natural forces which make for purity, and beyond the possibility of tillage which assists Nature to accomplish her beneficent work in the interest of man.

It is only a well-tilled humus that can satisfactorily deal with excreta. If there be an excess of water, *i.e.* if the humus is drowned, as in a sewage farm, then any protective action is very problematical.

The following is of interest in this connection.

1894. *High Wycombe* (Dr. S. W. Wheaton).—Town in a long valley between chalk hills. Rapidly increasing, population 15,000 (?). Lies on gravel bed, water mainly from shallow wells, partly from waterworks (private company). Town was sewered in 1882. The sewage runs to a twelve-acre sewage farm about a mile from the town, and “after mixing with a preparation of aluminoferric oxide” in a sludge pit, its liquid effluent flows directly on to the land. The water-closets of the town bad, and seldom adequately flushed.

Enteric fever had occurred every year since 1886. In one house the admission of a girl with enteric was followed by eight cases, and it was found that (*inter alia*) the soil-pipe of the closet passed over the well and dripped into it.

Again in December, 1894, after a heavy downpour, excreta were washed out of the sewers and into the wells.

1895. *Wycombe Marsh* (Dr. G. S. Buchanan).—Two miles below High Wycombe, and having the sewage farm of High Wycombe at its upper end. Population 700 in 140 houses.

Excreta disposed of partly in privies and pails (the contents being used for gardens), and partly collected by hopper closets, inadequately flushed and run into soak-away cesspools.

In autumn of 1895, twenty-four cases of enteric in nineteen houses situated between two streams,—the “Back-water,” 195 feet above Ordnance datum, and the “Wye,” 185 feet above Ordnance datum, running parallel to the Back-water and a quarter of a mile from it.

The direction of the flow of ground water was from the Back-water to the Wye and beneath the majority of the implicated houses.

The Back-water runs through the High Wycombe sewage farm of twelve acres, and in fact drains it. Owing

to the fact that the sewers of High Wycombe are laid in the water-logged gravel, and that the pipes *necessarily* leak, an enormous quantity of "sewage," estimated at 2,000,000 *gallons per diem*, is poured on to the farm. The volume of the "back-water" is about doubled after passing through the "farm."

In September, 1895, a mill below the village which had been long untenanted was let, and a dam previously partly opened was closed. This stopped the flow in the streams of Wycombe Marsh, with the result that the sub-soil water became impregnated with the sewage of High Wycombe, and the wells of the village were more or less affected.

Sir R. Thorne-Thorne, in commenting upon this case, says that it is interesting, since "it illustrates a danger which may devolve on one community as the result of works carried out solely in the interests of another community." (See also Beverley, p. 70.)

The river Wye flows into the Thames at Bourne End.

N.B.—2,000,000 gallons of water per diem is the equivalent of seven inches of rain per diem on twelve acres of land, or 2555 inches or 212 feet per annum.

#### DEEP BURIAL.

Again, the deep burial of *fæces* will preserve them, and not help their destruction, which is what we want. Deep burial in a porous soil may not be without danger.

For illustrations of this danger arising from burying infected *fæces* *deeply*, I would allude to a valuable report made to the Local Government Board in 1877 by Dr. Ballard, on a prolonged outbreak of enteric at Ascot in 1873-4-5-6. The epidemic was a milk epidemic, but the main difficulty was to account for the specific infection of the dairyman's well.

Dr. Ballard points out that the water-supply of this district is (or was) from wells sunk into a "running

sand" saturated with water, lying immediately below the denser superficial sand.

The excreta of the first case which was imported into the district were buried "in a hole in the sand" (depth not stated), and further there was a leakage of the water-closet drains into the sand, and Dr. Ballard suggests that the infected material may have reached the dairyman's well by the "running sand." He puts this view forward as an hypothesis, and not as a proved fact, but the calmly considered notions of so careful and contemplative an observer are not to be neglected.

This Ascot outbreak of enteric, affecting 69 persons, mainly of the upper and middle classes, appears to have been started by the importation in 1873 of a case belonging to the well-known Marylebone milk epidemic of that year. This latter epidemic was due to a case in Buckinghamshire. Thus we see, in these days of rapid transit, how far infections may travel.

The Marylebone milk epidemic is a good instance of the wrong disposal of infected fæces, and the danger of mixing them with water.

1873. *Marylebone milk epidemic* (Mr. N. Radcliffe).—The cause of this epidemic was traced to a farm in Buckinghamshire. The dairy well had become polluted by long-continued soakage from a piggery, the soakage creeping along the foundations of a wall. Against this wall was the farm "ash heap," in which the evacuations of the farmer (who died of enteric) had been buried, and upon which were cast all the chamber slops of the sick man's room. This filthy mixture drained with the soakage from the piggery into the well.

It is necessary to repeat that if fæces are placed on to humus no antiseptics must be used. Such addition merely prevents the humus from doing its work. The unpopularity of town refuse with farmers is largely due to the antiseptics which have been mixed with it, and



which, to borrow a phrase used by an agriculturist, effectually "*kill the dung.*"

#### WELLS.

I believe that a well, by draining the soil, helps to maintain the purity of the soil ; and it is most important to remember that when water for household purposes is pumped from the soil, the slop water returned (allowance being made for evaporation) must always be less than the water pumped. There is, therefore, not much danger of getting the soil round the dwelling water-logged and unwholesome.

When, on the other hand, an artificial supply is brought into a low-lying district already sodden with filth, and when we give up pumping and proceed to soak the land with our new supply, the purification of such soil becomes a matter of increasing difficulty.

Purification of the soil is attained by tillage, and by allowing the humus to breathe, and not by drowning it.

At my cottage at Isleworth, where I have a quarter of an acre of land, I have my own well, and I feel certain it is a much safer supply than the public supply of the district.

The instances of pollution of private wells by leaking sewers and cesspools are innumerable, but these have been mostly small family epidemics, and there can be little doubt that on the whole there is safety in private wells,—absolute safety if the owner have common sense and will take a little trouble. Private wells in crowded cities must always be dangerous, because of the inevitable leaking sewer.

I ventured to express the opinion in 1892 that a shallow well *properly made*, and in the midst of wholesome surroundings, is a perfectly safe source of water, and it is gratifying to find that this opinion is shared by so eminent an authority as Prof. R. Koch. Koch ('Bacteriological Diagnosis of Cholera,' 1894) advocates the

Wells

raising of water from the subsoil by means of tube wells, which he maintains is a perfectly safe method. He says, "People are now everywhere endeavouring to perfect the supplying of water on a large scale to the highest possible degree, but they should direct their attention to the procuring of water on a small scale also, and seek to limit the spread of cholera to a minimum, so far as it depends on water, by improving wells in the manner I have indicated. Just in this respect a great deal still remains to be done."

In a report made to the Local Government Board in 1893, by Dr. Bruce Low, will be found a table which shows conclusively how large a measure of protection against enteric fever is afforded by the use of wells for the supply of water, even though those wells may be (as in many instances was the case) badly constructed and negligently kept. In the Gainsborough Rural Sanitary District it was found that in 41 villages, with a population of 13,063, in which the inhabitants drank well water, there were in four and a half years 25 notifications of enteric; whereas in 10 neighbouring villages, with a population of 5693, the inhabitants of which drank Trent water or canal water, the number of cases of enteric was 167. In the first group the incidence of enteric was 1.92 per 1000; in the second group it was 29.3.

It will be gathered that my sanitary faith consists in a firm belief (I do not know a single fact to the contrary) in the protection afforded to our water-supplies by a well-tilled humus. I believe that organic filth of all kinds should be kept upon the surface to nourish the humus, and not be placed beneath it to endanger the wells; and I believe that the great cause of the modern increase of enteric fever has been the water-closet, which has fouled and is still fouling our sources of water. In proportion as we have given up foul water-supplies and have gone further afield the mortality from enteric has lessened; but the sudden outbursts of late years have shown that we have "scotched the snake, not killed it," and it is im-

possible to see any finality or real safety in our sanitary arrangements so long as they are dominated by the idea of putting our filth out of sight below the humus instead of on it.

#### ENTERIC AND OVER-CROWDING.

The late Dr. Murchison was of opinion that over-crowding was not an important factor in the causation of enteric fever, and it is quite true that in comparison with typhus or smallpox, or other mainly air-borne contagion, over-crowding plays an insignificant part among the pre-disposing causes of the disease we are considering.

Nevertheless a reference to the Registrar-General's last Decennial Supplement (supplement to the Fifty-fifth Annual Report, 1895) will show (Table VI, p. 115) that enteric fever was most rife in the more crowded counties. Thus the average death-rate from enteric fever for the whole of England and Wales during the ten years 1881-90 was  $\cdot 20$  per 1000 persons living.

Eleven counties had an enteric death-rate higher than the average, viz. Lancashire, Durham, and Nottingham,  $\cdot 27$ ; North Riding and South Wales,  $\cdot 26$ ; East Riding,  $\cdot 24$ ; Northumberland,  $\cdot 23$ ; West Riding,  $\cdot 22$ ; Cheshire, Monmouth, and Hampshire,  $\cdot 21$ .

On the other hand, the thirteen counties having the lowest death-rate from enteric were Hereford,  $\cdot 06$ ; Rutland,  $\cdot 08$ ; Bedford,  $\cdot 09$ ; Wilts and Dorset,  $\cdot 10$ ; Somerset, Surrey, and Oxford,  $\cdot 11$ ; Suffolk and Berkshire,  $\cdot 12$ ; Sussex, Huntingdon, and Cumberland,  $\cdot 13$ .

Broadly speaking, one may say that the enteric death-rate was highest in the manufacturing counties and lowest in the agricultural counties.

Hampshire is the only county which apparently contradicts this statement, so that it may be well to say that the relatively high enteric death-rate in this county is largely due to the big seaport towns, with hospitals (civil, naval, and military) and asylums.

The reason for the relative excess of enteric in the more crowded districts is not far to seek. Not only is the pollution of rivers at its maximum in these counties, but the space round the dwellings is often so restricted that it is impossible to prevent the pollution of the air and soil by excremental matter.

The rural counties are constantly being threatened with various expensive so-called sanitary works. It is important, therefore, to bear in mind that these counties suffer less from enteric than the wealthy urban districts.

In over-crowded places, where people live night and day in what one may call excremental surroundings, there is no escape from contamination. It must be in air, soil, and water; in their food; on their cooking utensils and clothing; in short, everywhere. The only real remedy for this state of things is space round the dwelling. Great schemes of sewerage and water-supply have hitherto merely increased over-crowding. Our 224 millions of local debt has not abolished enteric, while in some places it appears to have increased tuberculosis and diphtheria.

As an instance of enteric in crowded areas, I cull the following from a report by Dr. Bruce Low (1896) on a northern town (Middlesbrough) where enteric is endemic, and at times epidemic.

The population of this town has grown in little more than half a century from 5000 to over 85,000. It lies on a flat near the sea, and is mainly occupied with the coal and iron trades. The area is (excluding foreshore and tidal water) some 2700 acres, and in the most crowded parts 10,000 people are congregated on fifty-five acres of land. Dr. Bruce Low says, "The older parts of the town are very closely built; . . . the backs of some of the houses are often shut in by outbuildings, privies, and the like—some [houses] in the *old* parts of the town are very bad, . . . in a dilapidated condition. Houses of this class let for as little as one shilling per week, and afford shelter for a very low class of the population. . . . The employment at the works attracts numbers of persons

of a shifting class from a distance. Many of these people are improvident, intemperate, and uncleanly in their habits. . . .

"When a high tide coincides with heavy rainfall, the sewers are unable to contain the accumulated sewage and storm water. As a consequence there is backing up and ultimate escape of diluted sewage from street and yard gullies, flooding some streets and the basements of houses."

The *middens* are bricked and not cemented, and "the ground below is often saturated with black and filthy fluid. . . . Occasionally a single midden receives the discharge from as many as six privies. These privies are often a short distance from back doors and back windows, and in numerous instances face the pantry window and are within a few feet of it." In one case the people complained that their windows could not be opened "owing to the abominable odours emitted by the midden. . . . In some cases between the backs of dwellings in parallel streets there is a double row of midden privies divided by a narrow back passage only four feet wide. . . . The contents of the privy-middens are said to be emptied once a fortnight. The wet filth and ashes are mixed and thrown out of the midden into a wheelbarrow, which is emptied out upon the macadamised surface of the front street, and . . . the contents lie in the street for a time while the fluid filth soaks away."

Ash closets provided with pans are also in use. They are emptied on an average twice a week, but the system is greatly misused by the householder. We read that the pans are sometimes dusted inside with carbolic powder. "The pan contents . . . are taken to the dépôt, and after rough articles such as tin cans, matting, and the like have been removed, most of the residue is put into trucks for removal to the rural districts. . . . There is a growing difficulty in getting rid of pan contents."

"The dirty habits of the lower classes [in Middlesbrough] also increase the dangers above indicated. Indeed, it was found that the largest number of enteric

fever cases occurred in those parts of the wards occupied by the roughest class of the population, people who took no care of their houses or their persons, and paid very little attention to the state of their food. Some of these persons sleep in unwholesome 'box-bed places,' which resemble cupboards partitioned off from the living room and ventilated only by a small aperture, which when opened often overlooks the midden a few feet away."

Dr. Bruce Low is inclined to think, as well he may, that the fever in this town is "indigenous fever fostered by unwholesome conditions pertaining to the town itself."

Quite different are the circumstances of the town of Bicester (Dr. Theodore Thomson, 1896), with some 3000 inhabitants on as many acres of ground, a straggling and old-fashioned town with a stationary population. Most of the houses have good gardens or yards, and there are vault privies, pan-closets, and a few water-closets.

Of the twenty-eight persons first attacked with enteric twenty-six obtained their water from Crockwell Spring, situated at a point where the town sewer describes a semicircle round it, having the spring in the middle at a distance of some eight feet. This encircling sewer was found to be broken and leaking.

I have brought the closely packed manufacturing town into juxtaposition with a country town for the sake of contrast. In a place like Middlesbrough, where people live surrounded by fæcal befoulment, there is no possibility of their condition being made better or worse by water-closets. Nothing can remedy such a state of things short of total destruction and reconstruction. Pending that, however, I think the *daily* removal of excreta should be aimed at, and the local authority should endeavour to acquire a tract of land to give an object lesson in the advantage of a really scientific treatment of excreta.

Dr. Murchison was of opinion that the danger of infection from enteric stools was increased by putre-

faction. The permitting of fæces to remain about the house for a week or fortnight, just long enough for putrefaction to attain its maximum, must be terribly hazardous. The model bye-laws of the Local Government Board permit privies within six feet of the back-door!

Is no correction for dangerous sanitary negligence ever to be tried with the individual? A gentleman is placed in the dock for not having his bicycle lamp lighted; is nothing to be done to the man who endangers his neighbour's health by swinish apathy? Many towns correct filthy language by fine; can nothing be tried against filthy acts, which are infinitely more dangerous?

In a small town like Bicester or in a village the problem is very different. If the inhabitants would abolish their water-closets, and with them all underground sewers and cesspools, and allow their slop-water and storm-water to run in open channels or filtration gutters, all risk of epidemics of enteric would end. In such a place the great need is properly organised and daily scavenging. Every receptacle for filth—dry closet, ash-bin, slop-gutters,—all should be cleaned and swept out *every day*, and the stuff put to its proper use on a spot of public ground where the people may receive an object lesson which should show how much it is to their own interest to be cleanly.

I have been at some pains to lay down what I consider to be the true lines for disposing of enteric excreta, and incidentally for disposing of putrescible filth generally. I am well aware that in big cities, where houses have no curtilage, the convenience of the water-closet will certainly override all other considerations. If any improvement is to take place on the lines I have indicated, such improvement must begin at the outskirts of towns and not in the centre. But the water-closet is practically established by law, and no encouragement is given, even in country places, to the householder who may think as I do, and who may wish, for his own health and profit,

to work out his sanitary salvation on his own premises and independently of the sanitary authority. No man, of course, must be allowed to endanger the health of his fellow-man ; but we all do this when we poke excrement into a sewer without any certain knowledge that it will not leak to our neighbour's water. Individual responsibility in sanitation no longer exists, and it is impossible to fix any responsibility on public bodies.

The long list of epidemics caused by public works must make us think that this system is not working quite satisfactorily. In my published writings I deal with these questions at some length, and I also try to work out some of the details for the attainment of a profitable and safe sanitary independence. In opening this debate I have found it necessary to adhere strictly to the discussion of principles.

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## APPENDIX OF CASES.

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1867. *Guildford* (Dr. George Buchanan).—250 cases due to pollution of a new chalk well supplying part of the town, by quantities of sewage leaking from an old ill-constructed sewer.

Dr. Buchanan alludes to the fact that the conversion of privies into water-closets was a danger especially in the chalky soil of Guildford.

Sir J. Simon commenting on the case says, "The new well, no one could doubt, was most dangerously situated ; in the porous and fissured chalk stratum it was within ten feet of various sewers, one of which indeed was traversed by the iron delivery pipe of the high service," &c.



1870. *Warwick* (Dr. Buchanan).—"The public water-supply of the town scandalously filthy."

1873. *Sherborne* (see p. 76).

1870-73.—Mr. Simon gives a list of 147 places infected in these years. In almost all of these reports the words "polluted water" or their equivalents appear, but the details are seldom given, and no further analysis would prove profitable.

1874. *Lewes* (Dr. Thorne Thorne).—450 cases, due to pollution in the course of and at the periphery of water service caused by intermissions in the supply.

The water main at one spot passed through the centre of one of the public sewers. "This sewer was in consequence opened up, and when the arch of the culvert was removed at the point where the water main passed through it a jet of water suddenly shot up into the air. There was a hole in that portion of the water main which was inside the sewer."

1876. *Tideswell, Derbyshire* (Dr. Thorne).—Outbreak of enteric. "Spread of disease favoured by conditions in an intermitting water service allowing of suction of foul air into water-pipes."

1878. *Dewsbury District* (Dr. Thorne).—Population 124,286. Excessive mortality, especially enteric.

"Water-supply for some districts liable to pollution at its sources, and periodically fouled in the delivery mains during intermissions in the service."

1879. *Okehampton* (Dr. Blaxall).—"Water-supply exposed to pollution by direct communication between water mains and closets."

1879. *Redhill and Caterham* (see p. 69).

1880. *Enfield* (Dr. Parsons).—Occasional occurrence of enteric. Causes multiple. “Probable local contamination of intermittent water service by reflux of foul matters from water-closet.”

1880. *Haverfordwest* (Dr. Parsons).—Epidemic of enteric. “Public water-supply insufficient and liable to contamination both at reservoirs and in course of delivery by sewer air sucked in during intermissions through leaky flush-valves.”

1880. *West Cowes* (Dr. Ballard).—Enteric fever prevalent. “Intermittent water-supply liable to pollution in the mains.”

1880. *Sandown* (Dr. Ballard).—“Water-supply from waterworks taken from a stream much polluted in its course by sewage and excrement.”

1880. *Ventnor* (Dr. Ballard).—“Water-supply from waterworks intermittent, and polluted dangerously by free admission of sewer air into reservoir by means of overflow pipe.”

1880. *Llanelly* (Dr. Parsons).—“Water-supply constant and plentiful, but liable to contamination by filth of population living above intake.”

1880. *Newlyn East, Cornwall* (Dr. Ballard).—“Very scanty supply of water, and mainly from a well with which the village drain freely communicated near its outlet.”

1880. *Totnes* (Dr. Parsons).—“Outbreak apparently due to failure of town water-supply in the exceptionally dry summer, and consequent want of flushing of sewers, drains, and w.c.’s.

1881. *Blackburn* (Dr. Airy).—238 cases due to fouling of a culvert belonging to the water company by soakage from privies and surface drainage of adjacent cottages at the village of Guide, in which there had been cases of enteric fever.

1881. *Bodmin* (Dr. Parsons).—Severe epidemic. Cause? Water-supply liable to contamination. "Possibility of reflux of foul matters into public water-supply from closets flushed direct from an intermittent service."

1882. *Bangor* (Dr. Barry).—548 cases caused by the filter beds being flooded with raw river water containing excremental matter.

1884. *Sheerness* (Mr. John Spear).—"Water service intermittent, water mains often laid in same trench with tributary sewers, and interchange of contents between house drains and house service-pipes several times discovered."

1885. *Kidderminster* (Dr. Parsons).—1200 cases at least. Probably due to in-suctions of impurity during intermissions. The fouling of a deep well by percolation of subsoil water and sewage during a drought also not by any means excluded.

1883. *Hitchin* (Mr. W. H. Power).—"Outbreak of enteric associated with defects of the public waterworks, permitting occasional back-flow of sewage-polluted river water into the pumping well."

1884. *Beverley* (Dr. David Page).—231 cases due to contaminated supply of Beverley Waterworks Company derived from deep chalk wells (333 feet in all). Company's well and reservoir close to sewage-irrigated field belonging to East Riding County Lunatic Asylum. Sewage from asylum is supposed to have trickled alongside the well shaft.

1885. *Faldingworth* (Dr. Gresswell).—Small outbreak traceable to pump water polluted by washings from a fever case imported from Newark.

1887. *Margate* (Dr. Page).—Increase of mortality from enteric. "Water-supply pumped from a well in the chalk, on the outskirts of the borough beside a populous and growing neighbourhood. Water of bad quality and exposed to contamination by soakage of sea water and from cesspools."

1888. *Mountain Ash* (see p. 74).

1888. *Buckingham* (Dr. Parsons).—Sudden outbreak of enteric, confined at first to a poor suburb of the town, and specially affecting persons drinking water from a particular "spout." The water conduit to this spout exposed to pollution from a leaky drain which had received specifically infected excreta from a previous case of enteric fever.

1889. *Longton, Staffordshire* (Mr. Spear).—155 cases. "Special incidences of primary invasion in part of the town getting a supply from a particular section of the public water service."

1889. *Rochester* (Mr. Spear).—Public water-supply from wells in the chalk. "In very many cases at Strood, and in a few in Rochester, I found water-closets supplied direct from the mains, *i. e.* without the intervention of any cistern or tank. The danger of in-suction of air, and even of solid matter into the water-pipes from closet pans, during temporary discontinuance of water pressure, is well known to attend this objectionable arrangement."

1889. *Houghton-le-Spring* (see p. 70).

1890-91. *Tees Valley* (Dr. Barry).—1463 cases due to public water-supplies taken from the Tees, which was more than ordinarily polluted after a flood.

The water-supplies were owned by the Darlington Corporation and the Middlesbrough Water Board. "The river is found to be at all times subject to conditions of the gravest fouling by reason of the fact that human excreta and other filth are knowingly and deliberately conveyed to it."

1891. *Rotherham, Rawmarsh, and Masborough* (Dr. Theodore Thomson).—211 cases. Caused by the pollution of the High Level Water-supply of the Rotherham Corporation.

Pollution caused by the washings of manure inclusive of human excrement into the streams contributing to the public supply.

1891-2. *King's Lynn* (Dr. Bruce Low).—226 cases, mainly in February and March, 1892, due to fouling of public water-supply by the direct inflow of manurial filth inclusive of human excreta and typhoid excreta after a sudden thaw and flood.

1890-93. *Newark* (Dr. Bruce Low).—"Out of a total of 297 cases of enteric fever, 78·5 per cent. occurred among that half of the population which habitually consumed water from the public service."

This water consisted in part of raw unfiltered water from the Trent.

1892-3. *Chester-le-Street* (Dr. Maclean Wilson).—58 cases, mainly occurring in the area of the Consett Water Company, which obtained its water from the Stanley Burn. The burn was more or less fouled with excrement, but three miles above intake was a cottage draining into the burn, and which, in October, 1892, contained four cases of enteric.

The Consett water, before distribution, was subjected to sand filtration, but it is pointed out that the sand was obtained from the banks of the polluted river Wear.

1893. *Atherstone, Warwick* (Dr. Wheaton).—Outbreak of enteric due to the introduction into the town water mains of polluted water (particulars not given).

1893. *Worthing* (Dr. Theodore Thomson).—1411 cases in Worthing, West Worthing, and West Tarring. The epidemic in Worthing caused by leakage of a sewer into a heading run from the bottom of a deep chalk well. The epidemic in West Tarring and West Worthing presumably due to infection of the water mains by the entrance of filth from the surface through ball hydrants.

Speaking of the Worthing epidemic, Sir R. Thorne Thorne says, "Chemistry had all along failed to detect in the water any definite impurity, and it was only in the later stages of the epidemic that the results of bacterioscopic examinations went to furnish conclusive evidence, not only of its being fouled by fæcal matter, but of its contamination by the specific material of enteric fever."

Both waterworks and sewers the property of the Worthing Corporation.

1880. *Newport, Isle of Wight* (Dr. Ballard).—"Water-supply from waterworks good and abundant" (*vide* 1894).

1894. *Newport, Isle of Wight, with Parkhurst Barracks Prison* (Dr. Theodore Thomson).—516 cases (population over 10,000). 4·2 per cent. of those drinking "Newport water" were attacked; 1·7 per cent. of those drinking from private wells.

The water was from chalk wells imperfectly lined, polluted by an adjacent pond, and probably by more distant leaking cesspools, of which there were many. Water found to contain *Bacillus coli*.

1895. *Raunds, Northants* (Dr. Bruce Low).—129 cases, mainly traceable to two public wells which had been distinctly infected by the rinsings and washings of utensils and linen and stools of enteric patients.

PROFESSOR SIR WILLIAM T. GAIRDNER, K.C.B.

Any remarks that I can make on this subject will be mainly historical, for it is now many years since I was directly associated with sanitary work; and since it has become the practice to relegate typhoid patients to special hospitals I rarely see a case in our general hospital, and only now and then in private practice. I cannot but recall that the first occasion on which I ever opened my lips before this Society was when Dr. Murchison first brought before the Society the early sketch of the great work which he subsequently published on the continued fevers. Dr. Murchison was probably even at that time quite awake to the possibility of water contamination, but the doctrine of the day was not water contamination, but sewer gas, the details of which as a cause of typhoid fever he had had occasion to report upon, and very elaborately to work out, in connection with the Windsor epidemic.

At that time very few houses in Glasgow or anywhere else had the drains properly and regularly inspected, and when an individual case or cases of fever occurred, it was almost always possible to discover some leakage which gave a colour of probability to the theory that the escape of sewer gas into the houses might have been the cause of the fever in that instance. Thus the theory of sewer gas came to occupy what was no doubt an undue place in the popular estimation as a cause of typhoid fever; and many were the cases which were attributed to that cause, on evidence short of scientific exactitude. It was about this time that the illness of the Prince of Wales occurred, and gave an enormous impulse to the reigning theory; the whole search then was in the direction of sewer gas,

and the water-supply was not so much considered. More recently again water contamination attained much greater prominence, but it was found not to explain all cases of typhoid fever occurring in an endemic form. I am not sufficiently conversant with the facts to discuss what Dr. Poore has said about the prevalence of typhoid fever in Glasgow as compared with other places; but I am safe in saying that any prevalence of fever in Glasgow at all would be a difficult matter to explain, had it not been for the facts brought to light in connection with milk contamination. In fact (speaking of a time say twenty years ago), whenever typhoid fever occurred to any considerable extent, the tendency always was to look to the water-supply of the locality affected; whereas now we always in Glasgow tend to look to the milk, the purity of the water-supply being practically above suspicion. The real source of contamination is therefore mostly outside the city area, but the medical officer of health is always very careful to follow out the history of these contaminations, and he has been very active in obtaining legislation to check this evil by placing all dairies which supply milk to Glasgow, however distant, under careful inspection. I cannot refrain from bearing my testimony to the care and patience under difficult circumstances with which these epidemics have been tracked out by our sanitary officers. I remember when the question of milk contamination first came up, I was called in consultation to a town some twenty miles out of Glasgow, and when talking the matter over with the local medical man (not a sanitary official), there being at the time an epidemic of typhoid in the place, he said he knew all about it, and could confidently point to certain houses where typhoid fever would be sure to occur, because they got their milk from one particular dairy. He said "As soon as I observe the milk cart of A going to a door, I expect there will be fever in that house." I then asked him why he did not turn this knowledge to practical account; but he explained that it would be very



difficult to prove his case legally, and he would expose himself to the risk of an action for damages. Now this is just what the public officers have succeeded in doing for the protection of the community. They hold the wires and communications, and they can bring home the responsibility in spite of every effort to suppress the truth. I remember on one occasion some cases of typhoid occurring among our students with more than one death, and I appealed to the medical officer of health for an explanation. He said that his facts were not as yet sufficiently worked out for him to tell me definitely whence it originated; but he suggested that the students should abstain from taking milk from a particular source. He said, "Don't refer to me as an authority, and don't give any reasons, but by all means stop that milk." I acted accordingly, and gave instructions at the University restaurant by all means to issue no more of the suspected article. When this was done the epidemic promptly subsided, and the full and elaborate inquiry published a few weeks afterwards contained ample evidence that the whole epidemic then prevalent in the west end of Glasgow owed its origin to milk contamination from this particular source of supply.

#### SIR RICHARD THORNE.

The author has dealt with the subject from so many points of view, that it is difficult to formulate ideas upon it that would be worthy of submission to the Society. But I probably do not err if I say, generally, that the principal evils which he puts forward are sewers, water-closets, and public water-supplies in relation to typhoid fever in its epidemic form; and that his remedies are dry closets, the application of excreta to the surface soil, and shallow wells. Now no one, I imagine, will deny that the evils to which he has called atten-

tion have led to a great deal of mischief in this country ; but the inferences drawn with regard to them require a moment's consideration. Our great works of sewerage and public water-supply have been going on during the past forty years. We are not in possession of exact data over the whole of this period as to mortality returns, because for part of the time typhoid fever was not differentiated from typhus. During a considerable portion of the period, however, commencing just when the largest amount of this work set in, we have definite data. The passing of the Public Health Act in 1875, and the previous Act of 1872, led almost at once to the expenditure of millions of pounds per annum on works of sewerage and water-supply. Now it so happens that whereas the typhoid fever death-rate of England and Wales per thousand living was 0·37 in the five years 1871-5, there has been a constant fall since then, and the rate for 1891-5 was only 0·17. Synchronously, then, with these works, the value of which the author would call in question, we have had an enormous saving of life from enteric fever. The author very properly limits himself to the consideration of typhoid fever in epidemic form, but what we have to think of is what was the total amount of typhoid fever before the date when these works set in. The disease was then mainly general and endemic, whereas now we mainly meet with occasional but epidemic outbursts. Among other things, Dr. Poore, referring to the water-supply for the city of Glasgow, says in his summary, "it is an interesting fact that during the period 1881-90 Glasgow had a mortality from enteric fever slightly higher than England and Wales." It was higher, it is true, although it was only insignificantly so. But is the contrast which he makes a fair one? To compare Glasgow, a mighty city, with the whole of England and Wales, with thousands of villages and hamlets, must necessarily involve a fallacy. The true contrast lies between what Glasgow was before it got its new water-supply and what it has been since then.

The condition of Glasgow between 1865 and 1879 was that there were 510 deaths per million inhabitants every year from enteric fever, whereas the condition of Glasgow during the last ten years is that they have only had 193 per million. This surely represents an immense saving of life from this one disease during a period covered by the provision of the new water-supply. Incidentally the author has spoken of these great sanitary works in connection with cholera. Let us therefore contrast the period 1848-9, before these works were begun, with its 54,000 cholera deaths in a single epidemic, and the last epidemic period 1893-4 with only 135 such deaths, and this during the prevalence of a general European epidemic invasion, when England was exposed to a danger which was almost unique.

I will, however, frankly admit that the author is perfectly correct in saying that we now experience enteric fever in an epidemic form with sudden and alarming outbursts; but I maintain that the total amount of disease and death thus induced is small when compared with what it used to be. He has clearly pointed out how many of these outbreaks have been brought about by reason of public water-supplies. I believe that a large number of these epidemics have been due to faulty work—work of a character that is rapidly disappearing. Sewers were formerly laid down without the slightest regard to their leaking or not, or to their proximity to water-supplies, and the water-pipes were laid with joints well calculated to ensure the entrance into them of pollution from the soil around. But past wrongdoings in these senses do not justify us in condemning all public works of sewerage and water-supply. The author speaks very strongly as to the necessity of water companies being made responsible for their misdoings—a remark which I thoroughly endorse. But it is the public, through their representatives in Parliament, which is mainly responsible for the present state of affairs. The water companies get statutory powers by which they are, as it were,

whitewashed for almost every kind of mischief which their water may produce. Twenty-five years ago my predecessor, Sir John Simon, affirmed that "commercial water companies" should be held responsible for "certain sorts of malfeasance" in respect of "the fixed and finished parts of sanitary science." I have quoted his words in recent official reports, but the public take no more heed of them now than they did formerly.

I now come to some of the author's principal remedies. I gather that they are—firstly, dry closets, with daily removal, the contents to be utilised in the surface soil and "within the sight of the inhabitants." I would here point out that nearly all the author has said with regard to remedies applies, in my opinion, to strictly rural districts; whereas the examples he cites to show the need for the remedies are taken almost exclusively from large urban districts. He says, in his charmingly interesting little work on 'The Dwelling-house' just issued: "In London . . . we have neglected what I believe to be the most important of the principles of sanitation, viz. the keeping of organic refuse, whether solid or liquid, on the surface. The humus is the most perfect purifier," &c. Now I cannot conceive how we are to apply remedies such as he has mentioned to the existing state of affairs in London, and generally in our towns and cities. If we could go back a long way towards the pre-Adamic period, and begin over again with the author filling the post of an autocratic Baron Haussman, we might hope to have all he suggests carried out; but under existing circumstances I fear his remedies must be regarded as, in the main, impracticable, at any rate in respect of large towns; and as to the need of them, the fact remains that during the period covering the works which meet with his disapproval we have enormously diminished the mortality from enteric fever. Then, again, the author would evidently have us largely revert to shallow wells. But what would be the effect of this? It was the shallow well in our towns that was so largely associated with the

endemic form of typhoid which was the source of a mortality vastly in excess of that which obtains at present. Regrettable as are the occasional large outbreaks of epidemic typhoid fever in the present day, I think we should bitterly regret going back to the days of shallow wells. He further points out that if excreta be applied to the surface soil and properly tilled in, you get no mischief in regard to water-supplies, or otherwise. That I will admit if all he suggests could by any chance be carried out; but where are individuals and communities to find the soil which is to be well tilled? and how is the practice to be applied at a time when the people are leaving the country districts and are flocking into the large towns, where the population is consequently becoming denser and denser?

Although the author is doubtless quite right when he speaks on this subject, as a mere thesis, yet I do not think that nature made any provision for the disposal of specific excreta. Nature provides an easy means of dealing with the ordinary healthy excreta of healthy people living on the land; but dangers arising from specifically contaminated excreta are, I fear, much more difficult to be thus got rid of. Dr. Poore referred to Dr. Sidney Martin's experiments. The fact is that Dr. Sidney Martin has only commenced a series of experiments for the Medical Department of the Local Government Board, to ascertain how it is that enteric fever clings to certain towns and places, whilst it does not do so elsewhere; and he finds that by taking certain soils containing a certain amount of organic matter, that is to say, organically polluted soils, and then sterilising them, the enteric fever organism, when introduced into the soil, has already lived for 268 days; whereas in soil containing no organic matter, but similarly sterilised, it could only live for from fourteen to twenty-five days. Dr. Poore's criticism as to any deduction from the use of a sterilised soil, is only applicable to the first stage of an investigation still in progress. Both the soils in

question were sterilised in order that both could be treated as soils pure and simple. It was at first necessary to get rid of the extraneous organisms with which soil abounds, until we have learnt how far these may favour or interfere with the growth of the typhoid organism. But Dr. Robertson, of Sheffield, has recently announced, as the result of his experiments, that whilst in non-manured soils the typhoid organism completely disappeared in the winter months, yet in soils containing organic matter the typhoid bacilli were not only capable of surviving during the winter months, but of renewing their vitality and multiplying again on the advent of warm weather. He says no word about sterilising the soils. We may, therefore, justly raise the question whether it would be right to deal with specifically diseased excreta in the way suggested by the author. He says in his summary—and I agree with him—that burning is certainly the best mode of disposing of typhoid excreta. It has been a very common thing among medical practitioners, and also amongst those of us who are connected with the public health service, to advise people to bury specific excreta; but I am becoming very doubtful whether the mere disposal of such excreta in the soil is right, and I think I should now prefer to advise admixture with sawdust and burning with the aid of a little paraffin. But although commending the burning process, the author adds that these excreta might safely be placed on the surface of the soil, if well tilled. I cannot, in view of our present knowledge, feel that this would be unaccompanied with danger. I do not doubt the author's statement of the results he has obtained in what I may call a series of open-air rural laboratory experiments; but I do not think his conclusions are as yet applicable to ordinary communities under the condition of things obtaining to-day.

I trust that my remarks do not savour of hyper-criticism. That is an attitude I should studiously wish to avoid. My main object has been to show that there

are two sides to this question ; one which he has set out, another which I have endeavoured to indicate.

In conclusion I must thank him for the very handsome terms in which he has spoken of the work of the staff of the Medical Department of the Local Government Board, over which I have, at present, the honour to preside.

### PROFESSOR BOYCE.

#### *The necessity of systematic bacteriological investigation in relation to public health.*

In the prevention of typhoid there are two aspects, which I will not dwell upon, as they are universally recognised, viz.

1. *Accurate diagnosis*.—In this connection, from my own experience I would strongly emphasise the serum test, but the test to be of value must be carried out in a properly equipped clinical or bacteriological laboratory.

2. *Isolation and disinfection*.—This side is fully looked after by the Medical Officer and his Inspectors.

I will proceed, therefore, at once to that side of prevention which turns upon, if not the saprophytic life of the typhoid bacillus, at all events upon its transportation. Data have accumulated to show that the *B. typhosus* may be present in—

1. Sewage and dust.
2. Water.
3. Milk, milk products, *i. e.* creams, butter, &c.
4. Shellfish and other raw foodstuffs.

My personal experience under these heads is comparatively recent, extending only over a period of three years in Liverpool ; but, short as it is, it is sufficient to enable me to sketch the lines of procedure any community may adopt in order to supervise bacteriologically the above possible channels of infection. I will commence with the sewage.

1. *The bacterioscopic examination of the sewage of the towns.*—To investigate constantly—

1. Output from dwellings ; especial supervision of the hospital output ; the post-mortem room output.

2. The intake at the sewage farm.

3. The output at the sewage farm.

It is of the greatest importance that that department of a town which has the supervision of the sewage should be in touch with the bacteriologist, and that there should be constant bacteriological supervision. By this means imperfect disinfection of excreta is detected. There is no doubt that infectious excreta leave both hospitals and houses in an imperfectly disinfected condition. Again, the efficacy of the means of sewage treatment at the farm is supervised, and the bacteriologist is consulted when it is a question of obtaining newer methods of sewage treatment. Further, a knowledge of the biology of the characteristic sewage organisms is obtained, and the presence and identification of the *B. typhosus* is rendered easier.

2. *The bacterioscopic investigation of all sources of drinking-water.*—To be efficacious this will entail the abolition of—

1. Tanks of all kinds for drinking purposes. These have been over and over again proved sources of infection.

2. Private wells not under public control.

It will necessitate taking the drinking-water directly from the main, just as we take gas and electricity. There is no practical reason against this. The bacteriological investigation will be then limited, as it is in my own case in Liverpool, to—

1. Supplies derived from the surface (watersheds).

a. Examination of water before filtration.

b. Immediately after filtration.

c. In supply-pipes, including mains, branches, dead ends, storage reservoirs, &c.



order to enter a plea for adding one more trained and expert mind to the various local bodies which look after our welfare. The engineer, chemist, and medical officer have each been added, and it is now time that the bacteriologist should come in.

DR. D. S. DAVIES.

While I fully recognise that occasional typhoid fever may originate in many ways, its epidemic transmission by water or milk is of so great importance, and I have passed through so recent an experience of milk-borne typhoid, that I will confine myself to this point alone.

*Causation by Milk-carriage.*

In Bristol, which is a large manufacturing city and port of 317,000 inhabitants, the fever rate is remarkably low, and for the past five years has averaged 9 per 100,000 in comparison with the thirty-three large towns, in which it has averaged during the same period 19 per 100,000. Clifton, which is a registration sub-district of Bristol, containing a population of 45,000, is still more remarkable for its habitual freedom from anything but very occasional enteric fever, much of which occurs after return from the autumn holidays, largely amongst persons who have contracted it on pleasure trips. The last occasion upon which it was epidemic in Clifton was in 1879, when a definitely traced milk outbreak of some 80 to 100 cases occurred in Redland, a district of Clifton, amongst the customers of a particular dairy.

On Thursday, October 21st, 1897, the occurrence of some definite cases of enteric fever led me to suspect that the "influenza" which was currently reported to be prevalent, might prove indeed to be typhoid. A circular letter produced a prompt response from very many medical men, and the Widal serum test, which steered a remarkably accurate and straight course through the ill-defined

2. Supplies from deep sources (wells).

a. Water in the well at rest.

b. Water in the well when being pumped.

Constant examinations upon these lines are yielding important results. It is possible to form a local water bacteriological flora. For instance, the water pumped up from the very deep wells is almost sterile, but the water at the house tap contains a certain number of organisms. The data which are being collected will tell us why these appear. From time to time a new organism has been found in great abundance in the tap water; observation will indicate the meaning of this. By these constant examinations we are enabled more surely to identify an intruder or to have our suspicions aroused by the advent of certain sewage forms. Isolated examinations, as too often made at present, are worthless.

3. *Bacterioscopic examination of milk and milk products.*—I have not so far succeeded in isolating in suspected cases the *B. typhosus*, although in one instance I separated a form approaching in many respects Eberth's bacillus. I have not, contrary to other observers, found the colon group common. In my experience they are rare. In two ice creams I found the *B. Zopfii* in overwhelming numbers. There is no doubt that a knowledge of the common flora of milk—and it has one—is, as in the case of water and sewage, of great importance. A very great deal more supervision is wanted in the case of milk and milk products (ice creams), and I am glad to say that in our city the medical officer is seeking for the necessary powers.

4. *Bacterioscopic examination of shellfish and preserved foods.*—The examination constantly of large quantities of shellfish have shown that they harbour very frequently members of the colon group, giving well-marked agglutination with serum.

I have mentioned these four channels of infection in



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STBURY.



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and as yet obscure cases, soon convinced me and most of my professional brethren that we were indeed face to face with a typhoid outbreak. Four days' assiduous work so completely convinced us of the accuracy of this surmise, and so definitely pointed to a particular milk as the vehicle of infection, that on Tuesday, October 26th, at 10 a.m., I definitely stopped this milk-supply from circulation, and the result has been the speedy decrease and practical disappearance of the epidemic.

The course of the infected milk distribution is graphically shown on the diagram.

1st. The milk from the infected farm K was consigned for delivery to the distributor in Bristol, by whose man it was met at the Suspension Bridge, and distributed direct from the churn to customers, without going to any local dairy. Any milk not used on the round was returned, however, to the branch dairy and sold to casual customers. This supply served during the holiday month of August and up to the 26th of September, when the schools reopened, two districts: (a) the high level district, bounded on the east by College Road, and on the south by Gloucester Road; (b) the low level district, extending from the Mall to Grenville Place, Cumberland Basin, on the south, and as far as Bellevue on the east. On the reassembling of the schools at the end of September, however, this supply was confined to the high level district. Some of the earliest cases, developing at the beginning of October, showed themselves on the low level round, and many early cases also occurred on the high level round, but the incidence here was mainly in the second week of October.

On the high level round 29 houses were supplied; 19 were attacked, and 71 cases resulted—a ratio of attacks to houses of 65·5 per cent.

On the low level round 28 houses were supplied; 12 were attacked, and 30 cases resulted—a ratio of 42·8 per cent.

Taken together, 57 houses were supplied, 31 were



attacked, and 104 cases resulted—a ratio of 54·8 per cent., or about 2 cases per house.

2nd. A milkman started with a pure milk-supply from Ashton, but at Bower Ashton he received a supplementary supply from K (the farm alluded to in the first section), consigned to Clifton, after receiving which he supplied milk, 2 cases of typhoid resulting, at Bower Ashton, then proceeded to Leigh Woods, where 6 more cases appeared in 2 houses supplied, and then entered Clifton. In the course of his Clifton round the following resulted:—39 houses were supplied, 15 were attacked; ratio of attacks to houses 38·4 per cent.; the total number of cases was 37, or not quite 1 case per house. In this case a large quantity of good milk was polluted with a comparatively small quantity of infected milk, and it will be noted that the attack rate upon houses supplied was smaller, and the number of cases per house less. In one school, however, no fewer than 13 cases resulted, and 2 were fatal.

3rd. The third and last implicated supply came originally from a farm at Westbury, to which no suspicion attaches, and this furnishes one of the most interesting features of the whole outbreak. The method of procedure adopted in working out the epidemic is this. As soon as suspicion attaches to a farm this is inspected, lists of the customers on the various rounds are obtained, the milkmen on implicated rounds are called to give evidence, the routes of each round are mapped out, and the lists and maps compared with the fever case-books compiled from the inquiries on notified cases. The milk from the Westbury farm is brought into Bristol on three rounds. Two of these rounds were found to be entirely innocent of cases, and the third round was (with the exception of the single case of a servant in one house, situated near to the dairy where the returned infected milk went, and from which occasional supplies were possibly obtained) also quite innocent of cases up to a certain point, but beyond this point cases began to occur with

marked frequency. These facts went a long way towards exonerating the farm itself as a source of infection. On comparing the mapped-out routes it was observed that at the very corner where this change from freedom to infection occurred, this milk round crossed that of the K supply (previously referred to), and we were prepared to receive the admission that at this point, on occasions when the milkman found the demands of one large college house had shortened his supply, he was in the habit of buying a supplementary supply from the other man, who carried the infected K milk. After this, cases commenced at the very next house supplied, and recurred with considerable frequency throughout the entire route. The following are the results obtained:

Before adding the infected milk—11 houses were supplied, 1 house attacked, or 9 per cent.; 1 case resulted.<sup>1</sup>

After adding the infected milk—16 houses were supplied, 8 attacked, or 50 per cent.; 22 cases resulted.

The explanation of this implication, which for some time seemed obscure, settles the question of causation.

All the persons concerned have admitted before witnesses the accuracy of the facts I here adduce.

In the aggregate, up to Sunday, 14th November, 1897, we find—

<i>Total Houses attacked.</i>	<i>Houses.</i>	<i>Cases.</i>
Houses attacked on regular rounds ... ..	55 ...	163
Houses attacked obtaining casual supplies from the branch to which infected milk was returned ...	20 ...	32
Houses attacked obtaining milk from dairymen not known to obtain infected supplies (no multiple cases in any such house) . . . . .	13 ...	13
	<hr/> 88 ...	<hr/> 208

I am thus able to account satisfactorily for 195 out of a total of 208, or 93·7 per cent. of the total cases; and

<sup>1</sup> This case was afterwards found to have been in the habit of obtaining supplementary supplies from the infected dairy.



when it is remembered that the infected milk was supplied to one retail shop, round which a cluster of cases has occurred, and to one refreshment-room, and when the chances of infection by visiting are taken into account, the percentage of failure, which I hope further inquiry will reduce, is very small. The number of multiple attacks in houses upon the infected supplies is very notable. The largest number in any one house (a school-house) was 29; in others 11, 9, 7, 6, 6, 6, 5, 5, 5, 4, 4, 4, 3, 3, 3, 3, 3, 3, 3 cases occurred, and several double cases in other houses. No multiple cases occurred in any house to which an infected supply cannot be traced.

*Incidence upon the College Houses.*

Clifton College has suffered somewhat severely, but its experience has been amply confirmatory of the accuracy of my previous conclusions. Out of the many College "houses," containing large numbers of boys at susceptible ages, five houses only have been attacked, every one of which received, mixed or unmixed, the infected milk. In one house, receiving the unmixed infection (K's) during September, 29 cases resulted; in a second, receiving the same supply, six cases; in a third, which had apparently escaped up to the 24th October, it was found that it had not been put upon this supply until October 17th, and in due course two cases only followed, on the 24th and 27th October respectively (the lessened number of attacks furnishing an indication of diminution in the amount or intensity of the poison; only one out of eight houses was attacked of those supplied after this date, and only two cases resulted); in a fourth, on the Westbury round, only four cases resulted; and in the fifth two cases followed (on 24th and 27th October respectively) a single delivery of the infected milk (K's) on one Sunday early in October. The other houses, although situated amongst those attacked, and differing from them in no particulars of construction or occupa-

tion, but receiving an uninfected milk supply, were uniformly passed over. Each circumstance of the attacks was thus satisfactorily explained, and it became possible to advise with confidence that the work of the school should continue without interruption and without fear of further infection. Two college houses received the Westbury supply, one before, the other after the purchase of K milk; no case occurred in the first house, in the second four cases resulted.

#### *Individual Instances.*

Although individual instances are of slight value in establishing a case, they may prove useful as confirmatory evidence. In one case a lady attacked was the only one in the house who drank unboiled milk. In another case a child of nine on a day's visit to Clifton wished for tea at a restaurant, but being in a hurry to catch a train, drank some of the unboiled infected milk, and developed enteric fever at the end of a week (the incubation period in milk typhoid may be as short as five days). In another, a child visited an aunt who was servant at a house supplied with infected milk, where five other cases occurred. She drank milk, and sickened in seven days. In another case a child aged eleven, one of four children all living under similar conditions, and all using the same milk, but the only one who refused to have boiled milk with her bread and milk, was also the only one in the house attacked. In yet another case the only inmate of a house attacked was also the only one who drank unboiled milk. In a girls' school of 30 girls 11 were attacked. I am informed by the medical attendant that these all took unboiled milk for supper, but otherwise lived under the same conditions as the other girls. The remarkable immunity of the many schools in Clifton, none of which have been attacked except those few which were served by one or another of the infected supplies, is very significant. In one large girls' day school in Clifton, with

over 230 pupils, 10 girls, living in five houses, are absent with typhoid, every one of them in houses supplied with milk known to be infected. The pupils come from all parts of Clifton, and all the rest are perfectly free from the disease.

The last house supplied from the Westbury supply after infection was an institution containing 63 persons, of whom not one has been attacked. Inquiry shows that a system of scalding the milk in a steam-jacketed cylinder is in use at this institution, and the wisdom of this practice is justified by the result. It also supplies an answer to the oft-repeated question whether it is necessary for safety to raise milk to the boiling-point. Apparently scalding is a practically safe protection. Many individual instances of the immunity of those who only drank milk in tea, and the susceptibility of raw milk consumers, point to the same conclusion. Although there is now no infected milk distributed in Bristol, I have advised the continuance of the rule to boil or scald all milk or cream consumed, for the conditions at very many farms are no better than those disclosed during our inquiries, and with so many convalescents scattered over England from many infected towns, the danger of chance infection is ever present. The habit of drinking raw milk also appears to me to be essentially unclean. The percentage of houses attacked on each infected service is very heavy. In the Melbourne milk epidemic of 1879, reported by Dr. Allen, 23 houses were attacked, or only 24·7 per cent. of those supplied, and 40 cases resulted, with three deaths.

#### *The Conditions at the Farm.*<sup>1</sup>

The City Analyst reported :

October 30th, 1897. K Farm Pump and Stream above K Farm.—There is a remarkable closeness of agreement

<sup>1</sup> The cows on the farm were examined by a veterinary surgeon and pronounced healthy.

between these waters, which suggests that the pump is not supplied from a spring, but from the stream. Your inspection of the locality will have determined whether this is possible. The analytical results prove these samples to be highly charged with putrescent organic matter, whilst a direct microscopic examination, without the assistance of culture processes, shows them to be swarming with micro-organisms in great variety.<sup>1</sup>

An examination of the stream showed that it was utilised as the main sewer of the village of Long Ashton. We discovered that the sewage from 59 houses found its way into the Ashton brook. Of these houses, 37 are connected to a 6-inch sewer, which discharges into the brook about 125 yards below Yanley Lane, and the remaining 22 are connected to a cesspool in a field, the overflow from which passes directly down a ditch into the same brook, about a third of a mile above the sewer outfall. Other houses dispose of their drainage over the fields above the brook, whence in times of heavy rain it might easily be washed down into the brook, and one cottage at least on the brook discharges its sewage directly into it, and, thus highly charged with excremental matters, it flows directly through K farm.

The owners of property in the village, acting, I understand, under some pressure from the Rural District Council, appear to have been fairly active in connecting their houses with these drains, and in doing this the result has been to concentrate the excremental pollution upon the brook, while the Rural District Council itself has been at no pains to provide a sewer into which these drains might properly discharge, and have thus knowingly

<sup>1</sup> As the farm pump was only a few yards from the stream, its condition on analysis is easily explained. No illness of a typhoid nature could be traced at the farm or in the village of Long Ashton, but a man working on fields almost adjoining the stream suffered from an illness accompanied by diarrhoea during September, and his blood yielded to Dr. Klein a result indicative of typhoid on November 23rd. This man, no doubt, had "ambulant" typhoid, and polluted the stream by his dejecta. Heavy showers fell during the latter part of September.

permitted the persistent pollution of the stream running through important dairy farms. The cesspool to which I have alluded above is constructed of stone with defective joints, and the contents freely run away by a hedgeside ditch to the brook. I cannot learn that the cesspool is periodically or ever cleansed.

I have no reason to think that the farm in question is less than a type, as to its sanitary condition, including water-supply, of very many country farms, in which, therefore, the potentiality for wide-spread mischief is ever present, and the introduction of specific infective material the only element needful for disaster.

The city of Bristol draws its milk supplies from the three counties of Gloucestershire, Somersetshire, and Wiltshire, and of the 154 farms supplying the city, 85 are in Gloucestershire, 67 in Somersetshire, and 2 in Wiltshire.

The notorious apathy of rural district councils is in no one of these three counties controlled by a county medical officer of health, or by any special health machinery. Regulations for the control of our city dairies are made and enforced within our district, but we are, in common with other large communities, defenceless in regard to the immense number of milk supplies coming in from outside districts, and not under our control in any way. Wherein lies the remedy?

With regard to these districts, *it is not the making of fresh regulations that is needed, but the strict and universal and continued enforcement of the regulations that already exist.*

If, then, we can have no guarantee of effectual action on the part of any rural authority, or of any county council, for the altruism of these bodies does not appear to be elastic enough to include any consideration for the welfare or safety of alien districts, in what way can populous communities in our position best protect themselves?

There would seem to be no likelihood of legislative action by which each urban community might inspect,

register, and license outside dairy farms and farmers who furnish their supplies, to the exclusion of unlicensed places and persons.

But, in the absence of this, it would yet seem to be possible to take such voluntary private action, on the basis of the "Copenhagen Milk Supply Company" as shall ensure a reasonable and safe degree of protection.

The principle involved is that of a "controlled milk supply;" and they have, to this end, placed themselves, since their formation in 1878, under the independent control of experts and persons having no pecuniary interest in the business, but having the welfare and safeguarding of the public health at heart.

Under this system in Denmark, not only are very frequent analyses made (as many as 22,000 in a year), but all contractors are under a guarantee to abide by the rules, and such contractors only are employed; the cows on each farm are examined every fortnight by competent veterinary surgeons (seven in number), whose reports include the health conditions of the cattle and the condition of the cowhouses. An inspector also examines regularly and thoroughly the state of the farms, the conditions of milking, and the care of the utensils. Also a head dairymaid is employed to control the milking, with especial regard to cleanliness during the process of milking.

I am proposing to my health committee that a local committee of experts be formed on these lines, to include the necessary officers for inspection and control; and that the Dairymen's Association be invited to co-operate; that local dairymen be then invited to join the Association upon fixed charges, to be arranged so as only to just cover the necessary expenses of supervision; and in this way dairy farms complying with the necessary standard may receive the "sanction" of the control committee.

I have little doubt that, in their own interest, the best of the dairy producers and distributors in the city will gladly welcome the innovation; and that public

opinion will very properly boycott those who refuse or neglect to join. A public list of controlled dairymen should be kept posted in suitable places for the information of the public.

Any such scheme must of course be under the control of an independent and continuing committee;—it would be fatal to leave it to individual enterprise on the part of commercially interested persons, whose enthusiasm would inevitably become less and less under the pressure of competition, and the contemplation of dividends. The officers of the Health Department will gladly give their time and services in the organisation and arrangement of such a control service, though they cannot, of course, undertake the actual work of outside inspection. I trust that a sub-committee of the health committee will consent to serve upon the proposed "committee of control."

In this way, I believe, a practical and workable scheme of self-protection from typhoid or tubercular milk infection may readily become established.

*December 14th, 1897.*

DR. J. F. PAYNE.

The first question we ask ourselves in approaching this subject must be, Do we regard enteric fever as strictly a specific disease, or as disease possibly originating *de novo* in certain insanitary conditions? This point is fundamental. If it is a specific disease, so that one case of it can only originate from a previous case, then the problem of prevention becomes the problem of preventing infection, direct or indirect. If, on the other hand, there is any possibility of the disease being evolved out of some simpler form of disease, or of its bacillus being merely a specially virulent form of some harmless saprophyte, then the problem of prevention becomes a much wider one, involving not merely the medicine of the sick room, but

a large part of the science of sanitation, as applied to dwellings, towns, and so forth.

Without denying the possibility of the evolution of this disease, or its microbe, from some simpler condition or organic form, we may safely wait for future research to establish such a relation before we take account of it, and assume that the immense majority of cases of enteric fever, if not all, are derived from previous cases of the same disease. If so, just as in the diseases generally recognised as contagious—smallpox, scarlatina, &c.—each patient must be regarded as a centre of infection.

We have to consider in what manner infection is conveyed—by what channels it becomes dispersed. We agree that the infectious cause of the fever is constantly leaving the body of the sick person by various channels, but specially by intestinal discharges. Surely our most immediate and pressing duty is to attack and destroy this public enemy while it is under our hand and within our reach. There are, no doubt, difficulties connected with this method of combating the disease, and the bacillus may sometimes elude our vigilance. But these difficulties are nothing in comparison with those involved in the gigantic enterprise of searching for and destroying the bacillus in drains or sewers; in the putrid, stagnant waters of a gravelly soil; or in the enormous reservoirs of a water company.

Now let us suppose for a moment that it were possible so to manage every case of enteric fever in the country that every bacillus should be at once destroyed, and every particle of decomposing material disinfected before they leave the sick room. Can anyone doubt that the amount of this disease in the country would be very greatly diminished? On the strict specific view, the disease would be exterminated. But even if we allow a certain margin for a possible spontaneous occasional evolution of enteric fever, the result of extirpating all the living germs would be striking and decisive.

I would urge, therefore, that our first duty is to see to



this means of prevention, and to impress its immense importance not only upon the profession, but upon the public. Those who are in charge of typhoid patients occupy a position of grave responsibility; they are in the position of custodians of explosives or dangerous materials. On their vigilance may depend the life or death of persons living far away from the sick house. Let them take as a motto—"Prevention, like charity, begins at home." It certainly ought to be competent for medical officers of health to assure themselves that proper precautions of this kind are taken in every case of enteric fever, and, if need be, to intervene and see that they are made efficient.

I cannot, of course, now enter upon details connected with the disinfection of the discharges, clothing, bedding, &c., of the patient, but it may not be superfluous to notice a few points. With regard to the fæces, I assume that it is possible completely to destroy all bacilli which they may contain. No doubt the most perfect method is by burning, but it must be admitted that there are difficulties in applying this method when the dejections are mixed with much liquid, as is often the case. In private houses, at all events, it cannot always be satisfactorily done. The liquid method, mixing all dejections with a sufficient amount of perchloride of mercury and hydrochloric acid, seems satisfactory if thoroughly done, though merely sprinkling a little disinfectant over the substances, as used to be the custom, is evidently inadequate. What we want here is not any new method, but to impress upon all concerned the necessity of minute precautions, and also to insist that fæces are not the only channel of infection, but that urine, or even in some cases saliva and sputum, should be taken into account.

In regard to the disinfection of clothes and bedding, there are still some points of difficulty, especially with regard to woollen garments and blankets. Linen or cotton garments and sheets may, perhaps, be dipped in a

carbolic acid solution, which deodorises them at least, and the final disinfection is secured by boiling ; so that the risk of infection, if any, falls only on the laundress, and for various reasons is obviously not so great as in some other diseases. The use of the steam steriliser before washing obviates all risk. But it is not always recognised that these methods do not apply equally to woollen garments, flannels and blankets. These articles are never boiled by the laundress, since they would be injured by the process. I would point out, not in relation to this disease only, but as a general principle, that woollens may be sent again and again to the wash, and never be properly disinfected. I believe this is the reason of the extreme difficulty of keeping nursery flannels free from the bacteria which cause urinary decomposition. The same thing is true of the so-called natural wool undergarments now in vogue ; they are never properly disinfected: and this should be remembered in relation to daily personal hygiene as well as to disease.

We have, then, to be content with chemical disinfectants in solution, and possibly the customary carbolic acid solution, one in forty, may be sufficient, but one feels some doubt. There is great difficulty in the application of stronger chemical disinfectants. Lately at St. Thomas's Hospital, experiments have been made by Mr. White, our pharmacist, on the action of perchloride of mercury on woollens ; but it was found to stain and destroy the fabric so much that it could not be used.

In hospitals and public institutions the problem may, of course, be solved by passing all infected objects through the disinfecting oven or steam steriliser ; but in private houses that is evidently inapplicable. We still want, I think, some perfectly trustworthy method by which woollen articles can be thoroughly sterilised in the sick room or immediately outside it. When the illness is over, of course we can deal with bedding, &c., in a satisfactory way. It is of the *daily* precautions that I wish to speak. These precautions constitute our first line

of defence against the typhoid bacillus. The importance of maintaining it is shown by the occasional communication of the disease to nurses and others in the sick room. If this line of defence is once broken through, the enemy has a far wider area of mischievous activity. How it is to be met there is a problem I do not touch, not being able to discuss it with special knowledge. That question belongs to medical officers of health and sanitary specialists. The point on which I have ventured to dwell is one which concerns the whole medical profession. It does not seem too much to hope that were these precautions carried out with absolute rigour, and were their importance sufficiently impressed upon the public, the task of the health authorities and sanitary engineers would be rendered, as regards this special disease, almost superfluous, except for those rare and terrible accidents which sometimes occur, and by which even a single case of typhoid may give rise to a serious epidemic.

The great system of protection which has been built up by the labours of our sanitary reformers is of priceless value. Let us not under-estimate it, but in our domestic management of cases of typhoid let us act as though that second line of defence did not exist, and as if the prevention of typhoid depended on our domestic management alone.

#### SURGEON-GENERAL JAMIESON.

I have some diffidence in addressing this Society this evening, because my desire was to listen, not to speak,—to learn, not to teach ; but in the course of the discussion at the last meeting it occurred to be that some of our experiences in India and the colonies might prove interesting if they served no other purpose. From this chart you will readily observe the comparative prevalence of enteric fever at different stations.

In the army we are daily facing the problem of pre-

venting enteric fever. In India, for example, and in some colonies, notably Bermuda, we have to deal with it in its endemic aspect.

We have attacked and are attacking most of the channels which previous speakers have referred to as connected with the distribution of the disease; and I must confess that the outcome of our experience is that enteric fever has gained ground in some stations, although in others we have secured more or less permanent improvement. But what will probably interest Dr. Vivian Poore most is that it is in those places where we are obliged to dispose of excreta by the dry earth system, and where there is apt to be water-logging of the soil, that the disease has held its own against us.

In India, where this system is practically universally adopted, an effort is being made to dispose of all excreta by cremation, and this has been tried in some cantonments, where special cinerators, some on the closed retort, others on an open fire principle, have been in use.

One of our chief difficulties, however, even with a good cremation system, is how to deal with the flies which infest the neighbourhood of latrines and kitchens in warm climates. I would specially emphasise this matter, as flies are an agency in the distribution of enteric fever which none of the gentlemen who have taken part in the discussion have yet alluded to, and because they cannot lightly be disregarded by the advocate of a reversion to dry earth systems of sewage disposal. The part the flies play is now well recognised, and laboratory experiments, with which you are probably well acquainted, prove how dangerous an agency they may be in the conveyance of a disease such as enteric fever.

In this connection the sanitary condition of kitchens, especially in a country like India, is a matter of vast importance. The kindred subject of cholera prevention has, in the hands of Mr. Hankin, brought the Anglo-Indian to a full understanding of how unguarded, from the point of view of disease prevention, his kitchen ad-

ministration has been till recently. The very lively interest now being taken, I believe, by all classes in that country in what I may call their culinary surroundings, augurs well for radical reform in the near future. In barracks and hospitals much is being done to prevent flies entering or breeding in the kitchens, or indeed in any place where food is kept and stored, but the difficulties in the way of success are considerable. Professor Gairdner and Dr. Davies have specially drawn our attention to milk supplies. In some of our garrisons abroad milk is supplied from dairies under regimental or Government control; an experimental Government dairy being established, for example, at Umballa, under the supervision of an officer of the Army Veterinary Department.

These dairies are vast improvements on the methods of obtaining milk, to which the troops were accustomed not so very long ago; and they have been established mainly with the view of preventing enteric fever. But we are also attacking the milk supply at the "periphery," to borrow Dr. Poore's expression, and we hope gradually to see hospitals and canteens supplied with sterilisers of the Aymard type, so that only boiled milk may be issued to troops and their families.

We are, of course, also paying very careful attention to water-supplies, a very difficult question in many of our stations abroad. In many places it is practically impossible to secure a water-supply that is not liable to pollution. We are obliged, therefore, to seek means of destroying pathogenic organisms which are of practical, and not merely of theoretical utility.

Pasteur-Chamberland filters are being introduced in place of the old charcoal filters, but our experience of them so far is that, for military purposes at any rate, the Pasteur system of filtration is not very well adapted. We are, therefore, inclined to place our trust in boiling, and are experimenting with more than one apparatus which claims to supply boiled water ready cooled and aerated, without the delay experienced in obtaining these results

after boiling in ordinary kettles or open vessels. In India, a boiler invented by Mr. Larymore, superintendent of the Alipore Jail, is already in use in some stations.

Mr. Hankin recommended pinking all water-supplies, wells, tanks, filters, &c., with permanganate of potash as a preventive of enteric fever; and this has been consistently tried in India and elsewhere, but with conflicting results.

Quite recently Dr. Ghadi Ali, assistant in Mr. Hankin's laboratory in Agra, discovered a micrococcus which appears to have the power of destroying the enteric microbe and its allies. Where this micrococcus existed the Eberth-Gaffky bacillus could never be obtained, and laboratory experiments seemed to prove that the one, if it did not actually kill the other, at any rate prevented its being obtained by any of the ordinary culture methods.

During a severe outbreak of enteric fever at Agra last year the water-supply was experimentally inoculated with Ghadi Ali's micrococcus, and the epidemic ceased. Unfortunately for the experiment, but fortunately perhaps for those experimented upon, the water was at the same time boiled.

Prof. Boyce referred to the necessity of systematic bacteriological analysis. Our army, from the very nature of things, cannot carry bacteriological laboratories amongst its impedimenta, but we are fully aware of the valuable information they give us, and an effort is being made to have them established for the use of Army Medical Officers at our larger stations. Thus sanction has recently been obtained for equipping such laboratories at the military hospitals in Aldershot, Woolwich, and Dublin.

I have ventured to make these few remarks merely to indicate, to gentlemen taking an interest in this discussion, how much the department of the public service of which I have charge is aware of the vast importance of the subject, and how willing and ready we are to apply all

reasonable precautions for the prevention of enteric fever amongst our troops.

It may be of interest to refer to one or two points in connection with the above remarks, which have come under my own personal observation. In Bermuda, where enteric fever is most prevalent, the water supply is all rain water, collected chiefly from the roofs of houses and from open surface-collecting areas, and stored in tanks underground. As a rule each private house has its own tank. The tanks are excavated from the porous limestone, and rendered with cement. The chief danger of water pollution is therefore in the soil itself, and it becomes a question if the soil is not capable of cultivating the microbe. You have the following favourable conditions: a soft porous coralline limestone, proximity of sea level, and a hot sun. Until recently and from time immemorial the custom has been to dispose of all water by simply throwing it on the ground in the neighbourhood of habitations, the extreme porosity of the rock lapping up moisture like a sponge. In addition to this, the excreta have been disposed of by a system of cesspits and by burying, and the contents disappear in the common rock. It becomes, therefore, an interesting question if by this process the soil itself has not become planted with the seed of enteric fever. One fact is important, that with a hot moist and rainy season, from June to January, the conditions are then the most dangerous. It is a somewhat curious circumstance that New Providence, an island in the Bahama group, which has a geological structure identical with Bermuda and a similar water-tank system, has no enteric fever. I was there for four years, and I never saw a single case, although English troops were quartered there part of the time, and every winter there was a large influx of American visitors. Is it that the seed from an imported case has not yet been sown?

In India all experience points to the following facts:—  
1st. That the young soldiers and recent arrivals are sus-

ceptible to enteric fever beyond all others, and it is a remarkable fact that women and children suffer in a much less degree than men. 2nd. Cases at certain seasons occur at a large number of stations, occupying in the aggregate a vast area of country. 3rd. Such cases in a considerable number of instances are isolated ; I give, for example, two cases which occurred in my own experience. Two soldiers when on a drunken spree lay all night in a field in rainy weather ; both contracted enteric fever a few days afterwards. It seemed to me that the wetting so reduced the temperature of the body and its vitality as to render the men for the time unable to resist the pathogenic influences which are ever present throughout the length and breadth of India. 4th. In almost all the posts occupied by European troops co-operating with the various columns in Afghanistan, extending from the British territory up to Kabul and Kandahar, cases of enteric fever appeared, some of which posts were occupied probably for the first time since the world began. In the Nile expeditions also, and notably in the one in 1889 which I accompanied, it was found that the troops which went furthest up the river suffered on their return to Cairo in a far greater degree than the troops which endured less exposure and less fatigue, and it becomes difficult to resist evidence tending to show that in the causation of this disease there may be more factors than are generally acknowledged.

The influence of flies as a probable danger was forcibly brought to my attention in Egypt in this way.

Alexandria is a place in which enteric fever has been more or less prevalent ever since the British occupation of Egypt, and as a rule, but not always, it is more prevalent in Alexandria than in Cairo. Now immediately to the windward of the hospital, at Ras-el-tin, is a large accumulation of decaying weed, washed ashore by the prevailing winds. It attracts countless myriads of flies, which swarm in the wards, and in spite of all precautions they get into the milk, and infest everything containing moisture. In the



year 1892 the disease was unusually prevalent, and in endeavouring to trace the cause I made inquiries as to the disposal of the excreta. It was said to be removed daily to a place some miles in the desert, and buried. I caused a watch to be placed on the contractor, and the fact was elicited that the excreta were not taken to the desert, but simply deposited near the side of the road not a hundred yards from the barracks, and I think I was right in the assumption that the flies were on that occasion the cause of the dissemination of the disease.

#### PROFESSOR KANTHACK.

Personally, as a pathologist, I feel that I can contribute but little to this discussion, since during my observations I have been more concerned with the microscopical than the macroscopical aspects of typhoid fever, and can only deal with individual cases and not with epidemics. I agree with Dr. Payne that prevention should begin at home. In the prevention of typhoid fever there are two points to be considered: (1) the sources of the supply, *i. e.* whence the bacilli are poured forth; and (2) their distribution in space. With regard to the first point, until recently it has been considered that the bacilli are mostly found in the fæces, and that occasionally they have also been demonstrated in the urine. This is erroneous, and the question is more difficult than it looks, because one might be led to suppose that the bacilli were only present during the actual course of the disease or during convalescence. That, however, is a treacherous hypothesis. I may remind you that if an abscess appears during or after typhoid fever, or in suppurative osteitis, periostitis, or osteomyelitis, one finds the bacillus in many instances in the pus. Even more important is the fact that the bacillus has also been found in abscesses occurring months or even years after recovery from the disease,

when there can be no question of a re-infection. In almost every post-mortem on cases of typhoid fever in which they have been looked for, bacilli have been found in the bile or gall-bladder, even when there have been no morbid changes in the mucous membrane of the gall-bladder. Welch and others have shown that the typhoid bacillus has been found in the gall-bladder months and years after the patient has recovered from an attack, and that in the animal after an intra-venous inoculation it persists for a long time in the gall-bladder. There is one classical case especially in which cholecystitis was thus provoked and demonstrated some years after the fever. Now it is evident that if the bacillus can wander from the intestine into the gall-bladder, it may, after recovery, also wander from the gall-bladder into the intestine, so that a healthy and unsuspected individual may prove a source of contamination. Then the question may be raised as to how long the bacillus remains in the fæces after convalescence. We have no certain observations on this point, but judging from cholera, we know that in this disease after recovery the vibrio may be found in the fæces for a considerable time—a long time after they have become solid. It is therefore not safe to regard a typhoid patient as dangerous merely while he is ill, for after recovery he may still be a source of contamination or even of infection, and, what is more, this source may be altogether unsuspected. As mentioned above, if the bacilli can find their way into the gall-bladder from the intestine, there seems to be no reason why they should not find their way back into the fæces, so that a healthy man may still discharge bacilli from time to time. Whether, epidemiologically regarded, these are really points of actual importance I am not in a position to say, but we must abandon the idea that it is always possible to trace back the disease to an immediately preceding or recent case, although of course, for practical purposes, it may be assumed that there must have been a previous case giving birth to the bacilli. This, however,

I do not desire to discuss ; I merely wish to state facts which are frequently lost sight of or altogether disregarded. In respect of my second point the facts at our disposal are still very insufficient. Since the discovery by Elsner of a new medium for growing the bacillus (potato-gelatine and iodide of potassium) its study has been taken up with renewed vigour, and it is possible that the typhoid bacillus is more widely distributed in space than is generally assumed to be the case. I will, however, only allude to Lösener, who has for several years worked at the distribution of the typhoid bacillus, and by sceptics even must be considered a "fairly" competent person in this department of research. He has examined the superficial layers of soil in a field which had remained untilled for years, and there he found the bacillus. His cultures were subsequently examined by Pfeiffer and Kolle by means of their serum test, and declared to be the specific bacillus in question. He has also found this bacillus under conditions which, as far as he could judge, excluded the existence of typhoid fever. For instance, he inoculated a pig with the *Micrococcus tetragenus* ; it died and was buried in sand, and from its body he recovered the typhoid bacillus. It is, of course, open to anyone to say that this observer was not dealing with typhoid bacilli but with micro-organisms resembling them ; that, however, reduces the whole matter to an academical question, to which there is no answer. If we couple the above observations on soil with the experiments mentioned by Sir Richard Thorne Thorne which have been carried out by Dr. Sidney Martin, there is distinct evidence that the bacillus when once in the soil may thrive and remain dormant, ready, when the circumstances arise, to enter the water or milk supply under favourable conditions. It is necessary for us to make out the natural history of the bacillus in and out of the body, and to explore its usual haunts so as to discover its most vulnerable point. We must keep in mind that, whatever the starting-point of an epidemic or outbreak

may have been, the distribution of the typhoid bacillus may be wider than has been hitherto believed.

Now I must say a few words with respect to what is rather a sanitary question, which affects all personally and strikes nearer home, viz. the hygiene of the post-mortem room. I myself feel that the amount of disinfection carried out in the post-mortem room is scandalously little, and in saying this I do not exempt myself from blame. Everything is allowed to run down the sink, the intestines are held under the tap, and water is allowed to flow down them, and altogether it seems to me that almost every post-mortem room is a source of danger. I think we ought to apply the same rules to the post-mortem room that we employ in our own laboratories or in the wards.

I shall conclude with a few remarks regarding the control of the water-supply. I can only express my surprise at the small amount of supervision we seem to have over it. I was recently asked to examine the water in Cambridge, and on making inquiries I found there were two water-supplies, one from the chalk and the other from the lower greensand. On further inquiry I was unable to ascertain whether the two supplies were mixed or not, nor even whether there was a double service of pipes; in fact, there seemed to be no sort of municipal control over the supply at all. It is a matter for regret that such a condition of things should exist, viz. that the public health is left in the hands of public companies who can work quite uncontrolled, and that those who ought to control it are practically powerless.

#### DR. SIMS WOODHEAD.

With respect to the contamination of water-supplies, and without expressing any very definite opinion as to how they become contaminated, it has always been my feeling in connection with the putting in of cisterns

into houses—a much debated question—that the break in the continuity of the water-supply involved in the putting in of the cistern, although it may be a disadvantage to the occupants of the individual house, may be, and probably is, an advantage to the community at large. We have to do with two perfectly different and distinct conditions. On the one hand the cistern provides a break between the water main and any possible source of contamination, but on the other it offers or allows the presence of certain conditions of rest, pabulum, temperature, &c., which may be, and probably are, favourable to the existence of the typhoid bacillus should it gain access to the water in the cistern. It is now recognised that it is practically impossible to maintain a constant pressure in any mains, and therefore even with the best valve taps and the best methods of interfering with the reflux of water from the mouth of a pipe there must occasionally be a break-down in the available means of preventing the back flow of water. We must indeed acknowledge that high pressure and constant supply do not afford such a great safeguard as many people seem to think. Another point raised by Dr. Poore upon which I should like to insist very strongly, because I have recently had an opportunity of studying carefully the conditions by which in Poona the outbreak of plague was stemmed, is the question of early notification and inspection (*i. e.* before the second outbreak was arrested). The results in India were obtained almost entirely through a system of notification and inspection of patients and places. If we applied this system to our water-supplies, our sewage, and our diarrhoea and typhoid patients under the different conditions involved in the outbreaks of typhoid in this country, we should be able in many instances to prevent the spread of the disease which under existing circumstances too often continues to set us at defiance. We have had a good example of what can be done in this direction at Clifton, where by prompt and energetic measures the outbreak was practically cut down almost imme-

diately. In connection with that epidemic I may point out that when an outbreak is associated with the contamination of water, it runs a very different course from an outbreak associated with contamination of milk. In the former case it may sometimes be a very difficult matter, even when you have found the source of contamination, to eliminate that contamination; whereas in the case of milk Dr. Davies has demonstrated most clearly and admirably that by taking appropriate measures it is possible to check an epidemic at once, as was done at Clifton. In the case of water contamination, although the source has been traced here—of course a much more difficult matter to begin with, there may be such a sewage-soaked and infected condition of the soil, and there is such a possibility of overlooking sources of contamination, as at Worthing, that it may be impossible to say definitely that we have put our finger upon the exact or only spot by which typhoid contamination can enter. Even when the most elaborate precautions have been taken to get rid of known and suspected sources of contamination, unsuspected sources may nullify or undo much of the work that has been done. Therefore in looking at this question we are bound to acknowledge that, as regards infection by water, we have a much more difficult problem to deal with than when the infection takes place through the contamination of milk. We stand in need of further information in respect of the continuance of the typhoid bacillus in soil. In this I agree with Dr. Poore, Dr. Cartwright Wood, Dr. Sidney Martin, and the workers in the British Institute of Preventive Medicine, that the typhoid bacillus has a much greater chance of persistence in a water-logged soil containing a moderate amount of organic matter than in a dry, well-aërated soil, however large an amount of organic matter it may contain. In this, however, my actual experience is limited, though I hold a strong opinion on the subject. Then as to the constant examination of all possible sources of infection by both chemical and bacteriological

methods: Professor Boyce at the last meeting made out a very strong case, not for the value of isolated examinations, but for constant and regular examination so arranged that when there is the slightest deviation from the normal, those in charge of the public health might be put on their guard. That is, I believe, the great value not only of chemical, but also of bacteriological examinations. We cannot, as a rule, get much information from single examinations. In Manchester, under Dr. Delépine, practically the same system is at work as at Liverpool; so that, apart from the serum test for typhoid, the Medical Officer of Health is able to put his finger on outbreaks of disease very much more readily than was possible before,—not from the fact that he gets a typical typhoid bacillus from a particular case or a particular sample of water, but because he is put on his guard by the changed conditions which from time to time occur in the course of the examinations that are made on his behalf. One point should, I think, in this discussion be very strongly insisted upon, viz. the early information that can be obtained of an outbreak of typhoid by the serum test. It was noticed by Dr. Davies that at Clifton there were cases of supposed influenza which undoubtedly responded to the Widal test. It is now a matter of common observation, too, that outbreaks of diarrhoea frequently or even usually precede outbreaks of typhoid fever. If these cases of diarrhoea could be tested in connection with some of the outbreaks of typhoid, we should, I am convinced, find that a number, if not a very much larger proportion of such cases of “diarrhoea” were really mild or even severe typhoid cases, which appearing at an early stage of the epidemic, and before we are on the outlook for cases of typhoid, are overlooked or are never seen by medical men at all. In several institutions where they have had opportunities of observing the course of an outbreak, the officials have been satisfied that some of the cases at any rate which were supposed to be diarrhoea may really have been cases of typhoid. When we consider that the bacillus of typhoid

fever may remain in the intestine for some time after recovery as well as in the urine and gall-bladder, and when we also remember what Metschnikoff pointed out in relation to cholera, viz. that the vibrio may be present in the intestine a long time before an attack of cholera occurs, it would seem that we can have typhoid bacilli under very similar conditions present in the intestine in practically healthy individuals, and that this bacillus may remain in the intestine a considerable time before the intestinal tract becomes so altered that the bacillus has a chance of doing its special work. Then it is that bad, though not actually infected water, may help the typhoid bacillus to do its work. As regards what has been said concerning the persistence of the typhoid bacillus, it has always been my impression in connection with Welch's case and others that we have to do not with the persistence of the typhoid bacillus, but with reinfection. Eleven years really seems a rather long time for even a typhoid bacillus to remain in the body without doing any appreciable harm. On the other hand, we do know that under certain conditions reinfection may take place, but the constitution of the tissues may be so far altered that the organism does not, on the second occasion, react in the same way as it did on the occasion of the first infection. In times of epidemic I think all are agreed that the best way to deal with excreta is, if possible, to burn them, and that it is absolutely suicidal from our point of view to allow them to get into water. I, for one, should scarcely like to say that burial even in dry earth was, during an epidemic, the best way of getting rid of typhoid fæces. I believe that we may possibly over-estimate the danger of the presence of the typhoid bacillus in dry, well-aërated soil; but we cannot possibly over-estimate the danger of its presence in water-logged soil, especially when such soil contains a large amount of organic matter. At present we are not in a position to state by what organism or series of organisms the typhoid bacillus may be rendered inert, *i. e.* what organisms are capable of interfering with its growth and



multiplication, and what organisms will allow them to flourish. This, however, we may accept,—that it grows under very similar conditions to the colon bacillus, and wherever we have the colon bacillus we may expect under certain conditions to find the typhoid bacillus. If a favourable opportunity occurs, we may have a concurrent or simultaneous affection of the patient by active colon bacilli and by typhoid bacilli. It has been suggested that there is gradual transition from one into the other, but I think the explanation of many of such cases is rather to be found in the simultaneous action of diarrhœa and typhoid producing organisms, the former very often actually preparing the way for the latter.

DR. TIVY (CLIFTON).

I consider that influenza and enteric fever have been epidemic in Clifton during the months of October and November last, and this view was evidently taken by the Medical Officer of Health for Bristol, for in a circular sent me October 25th he states:—"Some obscure enteric fever appears to exist in Clifton, as well as some influenza with abdominal symptoms." The cases of influenza have been most puzzling, and many accompanied with severe gastro-intestinal trouble. In a number of cases seen by me the invasion was sudden, the temperature high—ranging from  $102^{\circ}$  to  $104^{\circ}$ ,—diarrhœa was present in several; there was, however, no splenic enlargement and no eruption, and all my cases convalesced in from eight to sixteen days. I did not have Widal's test used in any, feeling assured that the test is deceptive in cases unaccompanied by all the clinical symptoms of enteric fever, and this view is taken by the 'Lancet' of December 4th. Dr. Davies, Medical Officer of Health for Bristol, however, in his remarks before this Society on November 23rd, strongly advocated Widal's test as absolutely correct diagnostically in all doubtful cases, and mentioned that in

over 70 doubtful cases, examined by him in October and November, the diagnosis of enteric fever was made *certain* by it, much to the surprise of the medical men in attendance. Now, during the time these cases of influenza were about, a sudden and most serious type of enteric fever appeared, and in these, too, the invasion was sudden, the temperature high from the onset, running from  $103^{\circ}$  to  $105^{\circ}$ , delirium was common, severe pains in the limbs, diarrhoea was not present in all cases, the rose rash was not well marked, severe hæmorrhage occurred in many cases, and there was splenic enlargement; in fact, in the cases under my care I had no doubt they were enteric after the first week, therefore Widal's test was unnecessary. Dr. Davies has most cleverly tried to trace all the cases of both epidemics to the milk-supply of one small farm, having fifteen cows giving 100 quarts in the morning and 40 in the evening; this amount of milk is stated to supply sixty-five large houses, containing about 700 people, entirely with milk, and eighty-one large houses partially; out of this total, sixty-six houses, though taking the milk, were not attacked. Dr. Davies states that the cause of the outbreak was due entirely to rinsing the cans and tins, five in number, in impure water tainted with sewage; no allegation is made that water was added to the milk. On the other hand, the farmer, his wife, and daughter, who solely manage the dairy and have done so for years, positively affirm that they always, with their own hands, rinse their cans and tins in *boiling* pump water *only*, and that they never touch them with cold or unboiled water. The public analyst has not found typhoid bacilli in the samples taken the fourth week in October of the alleged infecting water or alleged infected milk, and no case of typhoid fever has been traced to the neighbourhood of the farm. It is said, however, that on November 20th, some seven weeks after the first of these cases was noticed, a labourer living near was found to be ailing, though he had worked every day as usual, and by Widal's test alone his blood was decided to be enteric.

I feel certain we have passed through two epidemics: one of influenza, in which the cases were numerous and many of them had abdominal symptoms; the second and most serious epidemic was one of severe enteric fever produced by sewer-gas poisoning owing to the entire absence of ventilation of the main drains of Clifton, and that thus, owing to a prolonged drought in August, September, and October, the drains were not flushed, and being ill-ventilated, sewer-gas in volumes was forced back, through over-pressure in the mains, through the street gratings and into the houses attacked, which stand on high ground and in close proximity to the large main sewer, which, after descending a steep declivity of about 270 feet, empties itself at all tides direct into the River Avon a short distance below the Suspension Bridge. The stinks of Clifton have been in the summer almost unbearable for years, and unless steps are taken at once to ventilate the main drains, and to divert the sewage of Bristol and Clifton entirely out of the Avon, it is much to be feared that the present visitation will be nothing to what will follow should a prolonged drought again occur.

MR. PERCY ADAMS (DEPUTY M.O.H. MAIDSTONE).

One of the most interesting features in the Maidstone epidemic, to which I would draw attention (as owing to the public inquiry to be held by the Local Government Board, my mouth is closed in respect of the incidents of the epidemic which has visited our town), is the precedence of diarrhœa before the epidemic itself began. I hand round a rough sketch showing how this was the case. We distinguish between the ordinary infantile diarrhœa and this "extraordinary" diarrhœa by marking the curves in different colours. I believe it is an accepted doctrine or opinion, that as a rule the incidence of autumnal diarrhœa is higher in males than in females. In Maidstone, however, the contrary obtained probably,

I would suggest, because the women drank more water than the men—in this case more polluted water. You will see on the chart that the curve rises gradually, and gradually merges into the epidemic curve proper; I may add that it will probably be an important feature in the official report which we shall issue, recording the history of this epidemic, because close to one of the places in which there was a possibility of specific pollution taking place there was a very heavy incidence of this diarrhoea. In the cottages close to a spring there was a history of diarrhoea from which nearly every member of the several families suffered (in some instances with symptoms very like typhoid fever). Subsequently, about the 14th or 15th of October, there was a case of typhoid notified in one of these cottages.

*January 11th, 1898.*

DR. EDWARD SEATON.

I have read the account of the exceedingly interesting discussion that has taken place on the prevention of typhoid, which has already ranged over nearly the whole subject.

In speaking a few words this evening, in answer to the invitation of Dr. Norman Moore, I do not of course attempt any review of the subject from the practical point of view of the Medical Officer of Health, but I will endeavour to supplement the discussion by a few words on certain points which may with advantage be emphasised.

In the first place let it be observed that there are certain epidemic diseases as to which the researches and teaching of the medical profession have imposed tremendous responsibility on sanitary authorities. Cholera, typhoid, smallpox, and typhus, are those which are generally regarded as pre-eminently preventable by the action of public authorities. But in considering their preventability from the practical point of view we cannot leave

out of consideration the state of medical sanitary organisation.

I have recently dwelt upon this subject in an address at the Sanitary Congress at Leeds, and I do not at the present moment wish to advert to it, except to repeat my conviction that it is a matter of the greatest importance, and one that deserves the consideration of all concerned, and especially of our own profession.

If we regard this subject of prevention from the point of view that typhoid is a disease which often arises otherwise than by water-borne infection, it becomes necessary to consider all available means of prevention.

This evening I would like to say a few words on the subsidiary question of notification of typhoid and disinfection. I am reminded in this connection that the system which requires compulsory notification of typhoid and other infectious diseases (with regard to which sanitary authorities have very heavy responsibilities) was inaugurated at Huddersfield, Bolton, and Nottingham almost simultaneously about twenty years ago. Much discussion took place at the time as to the necessity of including each of the diseases mentioned in the Schedule.

With regard to typhoid it was strongly urged that where the poison was intercepted and soil infection prevented, and where the material which was known to contain the poison could be subjected to sufficiently destructive processes, there the opportunity for limiting the diffusion of the disease would be greatly increased. The diminution of typhoid prevalence where notification and disinfection under skilled directions has been carried out has, I believe, fully justified the anticipations of the promoters of the system. It is quite evident that where hospitals are provided for the isolation of typhoid cases that cannot be properly attended to at home, all this work will be much more efficiently carried out than is otherwise possible, and that corresponding advantages will accrue to the public health.

Our knowledge with regard to methods of disinfection

has greatly advanced since then, and I may mention that I have received within the last two or three days a letter from my experienced colleague, Dr. Armstrong, Medical Officer of Health for Newcastle-on-Tyne, speaking of the great advantage that he anticipates from the use of sterilisers in enteric fever pavilions, which he believes will work just as well as they did at the Cholera Hospital of that city.

I believe that one of the urgent requisites at the present day for the prevention of enteric fever is the establishment everywhere of proper means for treating cases of the fever which cannot be attended to properly at home, and of effectually dealing with all infectious materials. I think that increased attention given to all the details of disinfection at hospitals will be a powerful agent in the reduction of typhoid.

Reverting to the advantages of notification, I may remind you that besides the opportunity that it affords of inquiry into the sanitary conditions of the premises where cases of typhoid occur, and besides, of course, sounding the first alarm in the case of water-spread epidemics, it may, if used with discretion (that is to say, if limited publicity be given), become a powerful incentive to increased activity in the way of sanitary improvements generally.

Since the Local Government Act of 1888 was passed, the authorities of several counties have inaugurated a system of circulating information with regard to the prevalence of typhoid in the component sanitary districts of the county. In some cases it has not gone beyond the circulation of information. I am not prepared to say but what that may be useful; but beyond this, in some counties the information is collected and edited by experts. Furthermore, where organisation allows of it, inquiry is instituted by the Medical Officer of the county into any small excess of the disease, and it is by means of such inquiries that some sanitary authorities are stimulated to carry out many valuable sanitary improvements.

We have lately at the Medical Officer of Health Society had under discussion the amendments that may possibly be introduced with advantage into the Notification Act; and amongst these is a suggestion that certain eminently preventable diseases—to wit, cholera and typhoid—should be notified at once to county authorities. It will occur to many that there is a good reason for this, inasmuch as many water companies supply water to a number of contiguous sanitary districts, and that consequently the information with regard to the prevalence in one district may be of importance to those in neighbouring districts.

So much, then, for the considerations which I think it desirable to bring before you from the medical officer's point of view. Concluding, I should like to say just a word upon the very large sanitary questions that are raised by Dr. Poore in his writings.

I think that Dr. Poore has done a very great public service in compelling people to think of what should be the ideals of sanitation in the country, as contrasted with those in towns.

In the days of Chadwick and Richardson we had ideal town systems placed before us, and there has been a tendency to think of the town systems as necessarily most applicable to the country. It may be, as Sir Richard Thorne has pointed out, almost impossible for us to reconsider the whole matter, inasmuch as we are committed to a large extent by the way in which towns and villages have grown up in the past, and are allowed to grow up at the present day. But public opinion may change, and a fair-sized kitchen garden, &c., may some day be regarded as a necessary attachment to a country cottage. We know that in rural France the same classes of people who live in clusters of houses in rural England are placed under conditions where they could carry out the systems somewhat similar to what Dr. Poore advocates. The existing state of things does not therefore prevent our considering the sanitation systems of

the country as something quite distinct from those of towns. I would say that when I have seen, as I often have to see, schemes for the drainage of a district, the combined drainage of little villages and clusters of houses by means of a network of sewers and drains spread over a vast unpopulated area on which could be placed a large and populous town, it seems to me that the ideas which originated with the venerated pioneer of sanitation, Sir Edwin Chadwick, have been much distorted, or rather I should say that a wrong ideal has come to be set.

Again, the carriage of the sewage to great distances and its treatment on large areas of land frequently involves a very heavy expense.

In some cases it is urged that these systems are absolutely inevitable, but latterly some hope is being afforded by the new bacterial systems of treatment, and it is to be further hoped that in the consideration of these systems and sanction of loans by authorities every opportunity will be given for improvement in this respect.

I would repeat in conclusion that we much value this exceedingly interesting debate which has been inaugurated so auspiciously by this Society, and I for one think it is likely to very materially assist in bringing about a further reduction of that typhoid fever death-rate which, as the medical officer for the Local Government Board has stated, has already been reduced something like 60 per cent. in our own country—a work of which we may all be proud. I thank you for allowing me to attend as a visitor, and for the attention you have given to these few brief remarks.

#### DR. CORFIELD.

I must join issue with the author in what he says with reference to the influence of the water-closet in disseminating disease. I say the great cause of the enormous decrease in enteric fever since 1875 has been



the increased adoption of the water-carriage system, and the increase in the number of water-closets in this country. Those parts of the country in which water-closets are least used, and the water-carriage system is the least perfect, are precisely the parts of the country in which enteric fever is most prevalent. London is, I suppose, the most perfect example among the large towns not only of this country, but of the world, of a completely water-closeted town, and of the water-carriage system. The mortality from typhoid fever in London between 1881 and 1890 was actually less than that of England and Wales generally. The counties showing the highest typhoid death-rates for the same period were Nottingham, Lancashire, Yorkshire, and Northumberland, in which one dry system or another is more or less in vogue, and water-closets are not much adopted. The same remark applies to South Wales in opposition to North Wales, the mortality from typhoid fever in the latter being less than three fifths of the former.

Bramah has been mentioned as the inventor of the water-closet, but it is far too beneficent an institution to have been invented by any one person. It was originally employed by the ancient Romans. Vitruvius describes it with its cistern, pipes, and taps, and remains thereof have been found in the palace of the Cæsars at Rome.

In a very remarkable little book called the 'Metamorphosis of Ajax' (1596) there are some very curious diagrams, from which you will see that, practically speaking, the apparatus had then been devised, though not exactly in the form in which we now use it. It was described and drawn by the author, Sir John Harrington. The first patentee of a water-closet in this country was not Bramah, but one Alexander Cummings, whose name I take the credit of having rescued from obscurity. His patent is dated 1776. Bramah modified it afterwards, and so it bore his name down to comparatively recent times. Now twenty-five years ago it was the generally received view of the profession, that the poison of enteric

fever could arise *de novo*, being produced, in fact, during the decomposition of excrement. That view has been gradually abandoned, it having been shown (see paper by the speaker in the 'Proceedings of the Epidemiological Society,' 1874) that towns might be for years in a condition suitable for the spread of enteric fever without the disease occurring until the introduction of a case set it going. That was one way of proving that it did not arise *de novo*. Another way in which that theory was gradually got rid of was by tracing out the source of cases which were supposed to have arisen *de novo*, but I need hardly point out that there are great difficulties in the way of carrying out such inquiries to a successful issue. I remember one instance in a large country house where there were twelve maid-servants sleeping in one long dormitory. One girl was attacked by enteric fever twelve or fourteen days after a sewer had been opened in the yard, emitting a very foul smell, which this girl had particularly noticed and complained of. I was assured that no case of enteric fever had occurred in the district for a long time, and really it appeared to be an excellent example of the *de novo* origin of the disease. On inquiry, however, it was not only shown where the disease came from, viz. from a cottage six miles away, but it was also shown how the infection had been conveyed into the sewer in question. It is generally admitted now that this disease is conveyed by water and uncooked foods—shell-fish, milk, water-cress, &c. It is not as well recognised that it may be communicated by contaminated air.

Dr. William Budd, whose book is still one of the best on this subject, strongly maintained this view, and I believe he was right. Sir William Gairdner referred the other night to the case of the Prince of Wales; that case ever since it was investigated has always been thought to have been a case of air-borne enteric fever from sewer gas escaping into the house where he was staying. On account of that remark it occurred to me to look up my notes of twenty-seven years since, which contain an

account of almost everything connected with that case and the house in which it occurred. I came to a conclusion that I did not expect to come to. I may say that at that time the supplies of water and milk were excluded. It was clearly shown that they were not the cause of the disease. It was therefore put down to the unhealthy condition of the house. It was stated in the press at the time that there were two cesspools under the house, and that the soil-pipe of the water-closet in the Prince's apartments discharged into a cesspool underneath, and that this soil-pipe was, moreover, not ventilated, there being a trap near the top of it. All these statements were entirely inaccurate. There was no cesspool under the house at all. Those who first examined the house for the press were, it is true, told that there were two cesspools in the basement; but Dipstone traps in Yorkshire are called cesspools, and these alleged cesspools were merely Dipstone traps into which sinks discharged. They were not connected with the water-closets at all, and the pipe from the closet in question was outside the house, and discharged into a branch drain outside. Lastly, the soil-pipe was fully ventilated, being carried right up full bore above the roof. There was a bend near the top to avoid some projection, but the bend could not possibly hold water, and it was this that the reporters took for a trap. At the same time it had to be admitted that the sanitary arrangements of the house were somewhat defective. On going through my notes, however, I have been forced to the conclusion that it was not a case in which the disease was conveyed by sewer air, because the persons who were most in the house did not get it. It affected solely the gentlemen visitors and male servants, and not the ladies or maid-servants of the household. I infer, therefore, that it was caused by some food-poisoning, possibly taken out of doors.

We have been told lately that the enteric fever organism has not been found in sewer air. If that organism is not in sewer air, I should beg leave to doubt

whether it is the cause of enteric fever. I am satisfied that the chief cause of enteric fever in London up to a recent date was the communication of the waste-pipes of cisterns with the drains and sewers, thus allowing sewer air to get access to the water. The number of instances of this kind which have come to my notice have thoroughly convinced me that such is the case. It has been hinted that we might do without cisterns, but I may point out that the water-supply has to be cut off from the mains from time to time, and it is indispensable that there should be some means of storing water. The removal of the cisterns from houses in East London was one of the causes of the water famine last year, and they are now being replaced.

Lastly, I wish to point out that there is a remarkable and hitherto little noticed difference between the spread of typhoid fever and cholera. I may illustrate my point by the city of Lyons, where cholera has never spread; indeed, it is the only large city where cholera has been introduced without showing a tendency to spread. When it has appeared in Paris or Marseilles, people have flocked to Lyons, taking cholera with them, and yet there has been no outbreak of the disease there on that account. Prof. Pettenkofer has written a treatise on the subject with the view of explaining how it is that cholera has never spread in the city of Lyons. On the other hand, enteric fever is endemic there. In fact, Lyons is a city remarkably prone to this disease. I have seen practically all the medical wards and half the surgical wards in the hospitals there full of cases of enteric fever. Therefore there must be circumstances in the city which favour the spread of enteric fever, and not that of cholera, though both are water-borne diseases. I believe this circumstance to be due to the fact that it is a city of cesspools, and that the soil, ground-air, and air in the houses are very largely contaminated by fæcal emanations, and that enteric fever in Lyons is more spread by foul air from this cause than by food or water. If it were spread by

water, then cholera would be spread in the same way. I think, therefore, that Lyons affords an example on a large scale of enteric fever being communicated by means of contaminated air.

DR. EDWARD SQUIRE.

Some twelve years ago, in bringing before this Society some points bearing on an outbreak of enteric fever amongst the troops in the Soudan, I gave reasons for supposing that the outbreak there was spread and the infection carried through the air by means of dust containing fine particles of the dried excreta of typhoid patients. The circumstances were peculiar in that the water-supply consisted exclusively of condensed water. I may mention that typhoid patients from the circumstances of the case were often compelled, either from necessity or from sheer pluck, to go on fulfilling their duties for the first one or two weeks. Their excreta had to be deposited in trenches or latrines, where under the influence of a very hot sun they were very soon dried and converted into powder. The observations of Martin and others, alluded to by Sir Richard Thorne, and especially those of Uffelmann and Germano with reference to the virulence of the typhoid bacillus remaining after desiccation, give some point and offer some support to my suggestion made twelve years ago. In this country that mode of spread will have very little influence, but in hot and dusty climates it is a point requiring more consideration than it has yet received. Seeing how many officers and young soldiers are annually lost in India and Egypt from this disease, I think this possible source of infection is well worthy of further consideration and more extended investigation.

## DR. FLETCHER LITTLE.

The first thing is universal notification, so ably dwelt upon by Dr. Seaton. It is necessary for us to ascertain the whereabouts of every case of infectious disease in the country, in order that they may be dealt with. When we notice that the mortality in the districts where notification exists is about one half that which obtains where the Notification Act does not apply, we are driven to the view that all cases should be notified. It will be necessary for the community to make up its mind to compensate the bread-winners of families who are in many instances prevented from attending to their work when there is typhoid fever in the house. We have had instances of this in my district at Harrow. Widal's or some like test should be placed by local authorities at the disposal of medical practitioners. The importance of this I need not dwell upon, because typhoid fever is often mistaken for influenza, and it is often difficult to distinguish it from phthisis. We have lately had a very sad instance of this difficulty of diagnosis in the case of a member of our own profession, who went about for some days with a temperature of over  $100^{\circ}$  because he was not recognised to be suffering from typhoid fever, which terminated fatally. Then, again, all public buildings, hospitals, parks, palaces, docks, railways, colleges, schools, churches, &c., should be directly under the care of the Medical Officer of Health for the district. At present we have large numbers of public buildings which are outside his control. At Dublin, for example, there were barracks where, whenever a fresh regiment arrived, a certain proportion of officers and men almost invariably contracted the disease and some died. My own feeling is that every hospital throughout the country should be examined and reported upon by the Medical Officers of Health. I may remind you of the fact that one of the largest hospitals in London was shown to have old-fashioned

drains containing sewer gas, yet until the Medical Officer of Health for the City was called in nothing was done to remedy an admittedly defective system. I would insist on the water test for all drains. When I was appointed to Harrow there was not a single drain plug in the place. It has been argued by some that it was for the best that drains should have joints made with puddled clay, but after a time I was able to show them that the ground all around them was saturated with sewage, and that the saturation of the soil with sewage, especially if it contained typhoid bacilli, was one of the great causes of the spread of the disease. Therefore I would insist upon the general application of the water test, the smoke test being altogether inadequate. As regards water-supplies, Dr. Poore suggested that the public authority should not have charge thereof, because, forsooth, the body that at present rules over London was too political in its aims. In order to have water abundant you must have it cheap. The West Middlesex Water Company pays 10 per cent., and the Chelsea Water Company 12 per cent. to the shareholders. The London County Council can borrow money at less than 3 per cent., and at the end of a generation or so, by means of a 1 per cent. sinking fund, the water supply would belong to the people of London. I think I ought to mention that at present it is almost impossible for a public authority to go in and examine the condition of a Water Company's property except as a trespasser. The municipality of Stockport has lately been endeavouring to get admission to its Company's grounds, but I am not sure whether they have yet been successful. The next point is that all sources of milk supply should be looked after, not that of some districts but all over the country. Much of the milk drunk in London comes from enormous distances,—from Derbyshire, for example, and from abroad ; it follows that the sanitary condition of the dairy farm in Derbyshire has a direct bearing on the sanitary condition of Marylebone. Now the railway

companies have a silly, almost wicked rule, that milk cans are not to be sealed up. Some time ago at Baker Street and Victoria stations I saw printed notices that Spiers and Pond were not responsible for the purity of the milk sold there,—that is to say, they were offering for sale milk of which they could not guarantee the purity. From much experience I believe that all raw shell-fish should be regarded as “suspect.” Raw oysters are one of the media through which typhoid is produced. We recently had a very good example of this at Harrow. Mussels have been found similarly to convey the disease, and this probably applies to all other kinds of raw shell-fish. Then, too, I think that all cases of typhoid should be treated in hospital,—with this reservation, that they should be attended by their own medical advisers. We are carrying out an experiment of this kind at Harrow, and I hope later on to be able to report to the Society thereon. Next I would suggest that Dr. Poore’s proposal that typhoid stools should be applied to the surface of flower-beds in public gardens—in Cavendish Square, for example—should not be carried out. If it were done, say in the neighbourhood of a dairy farm, pigeons and birds, &c., would soil themselves with this excreta, would deposit it on the roofs, and thus foul the rain water, which is often used to wash the vessels in which milk is sent to town.

DR. C. THEODORE WILLIAMS.

While the discussion has gone on about the question of bacteriology of typhoid fever and water-borne poisoning, we have heard nothing about the vulnerability of individuals. It is a curious fact that some families are distinctly vulnerable to typhoid fever, while others are not. What I want to know is how it is that some individuals are so distinctly liable to the disease, while others are less so. My impression is that certain races, too, are more prone to the disease than others,—the Anglo-Saxon race, for



example, being more prone to it than the Latin and Southern races. I remember on one occasion having taken the only room I could find vacant in Naples at the time of the carnival, and I had not been there long before a medical friend called upon me, who had made a special study of typhoid fever in that city. He insisted on my leaving the room, and afterwards told me that he had known seven cases of typhoid to occur in that very room, all Englishmen, five having died. We know, moreover, that there are certain hotels in Italy where you may generally predict that English people will get typhoid fever; and in spite of warnings they go to them nevertheless, and they, as a rule, contract the fever, while visitors of other nations do not become affected. A delegation of the foreign doctors at Naples at one time interviewed the authorities, to call attention to the fact that typhoid fever was very prevalent in a certain district of the city. The official who received them admitted the allegation, but pointed out that it was the strangers' quarter, and that was his ground for not interfering, as he had only to look after the health of the natives. At that time there were some 1200 wells, all more or less infected with sewage. There was no proper water-supply, and the drainage from the upper town gravitated into the lower part where the strangers lived, infecting the wells, and so typhoid fever prevailed. The entire neglect of sanitation by the Naples authorities brought about a terrible punishment when the cholera broke out in 1884, for the disease spread like wildfire, and no less than 8000 individuals, for the most part in the prime of life, fell victims. I know a town in Sicily, after Dr. Poore's own heart, situated on the side of a hill where a pure water-supply is obtained from an old Roman aqueduct which enters at the top of the town. In the course of conversation with the only medical man of the place I ascertained that there was no typhoid fever, and never had been any. I asked what about the sanitary arrangements, and he told me they had no

drains or closets of any kind. I asked him how they managed, and he told me to get up between six and seven one morning and see for myself. I did so, and I found, to my surprise, that this being a walled city, the women and children went out by one gate, and the men went out the other gate to relieve themselves; and they left it to the winds of heaven, the birds of the air, and other natural influences to do the rest.

#### DR. HORTON-SMITH.

How long after an attack of typhoid fever does a patient continue to be a source of infection? We have to ask ourselves the question whether in some cases typhoid bacilli may continue to be excreted in the urine or fæces for some considerable time after defervescence. This is a very important point, because if care is no longer being given to the urine and fæces, the disease may continue to be spread. Very few observations seem to have been made on this point so far. Probably in most cases the typhoid bacilli disappear from the excreta soon after, or even before the temperature has become normal. But in two cases the bacillus has been found in the fæces, in one 12 days and in the other 41 days after defervescence. In two other cases typhoid bacilli were found in the urine, in one 22 days and in the other 21 days after defervescence. Pending further observations I would suggest the precaution of taking the same extreme care of the fæces and urine during convalescence, until the patient actually leaves hospital, as during the fever itself.

Even after the bacilli disappear from the excreta they may still remain in bone marrow, setting up chronic osteomyelitis. In two cases they have been found, on operating, in the pus of sequestrum-containing cavities, *six years* after the typhoid fever, and in one of these cases there had been a sinus discharging this infected pus during the whole of this period. It is true that no

epidemic has ever been traced to such a case, but unless recognised it is without doubt possible that it might start one.

With reference to the advisability of placing the typhoid excreta on the soil, it should be remembered that it has been shown by Drs. Sidney Martin and Robertson that the typhoid bacillus can live and spread in some soils, whether previously sterilised or not. Lösener also has actually found the typhoid bacillus in the soil of a ploughed field.

It would seem, therefore, that under certain circumstances the typhoid bacillus can undoubtedly live as a saprophyte in the soil. Hence it would seem better to treat the excreta efficiently with antiseptics before placing them on the soil, or where feasible to destroy them by the petroleum-sawdust method as recommended by Sir Richard Thorne Thorne.

#### MR. H. E. DURHAM.

I take it that the prevention of enteric fever will probably always depend upon its identification, and the recognition of enteric fever will always go hand in hand with the recognition of the enteric or typhoid bacillus (or its doings). The evidence given, even by recent observers, of the discovery of the "typhoid" bacillus is often so inadequate, that I may perhaps be permitted to say a few words concerning its recognition. The most important point wherein the true typhoid bacillus differs from other bacilli which resemble it is its want of power to produce gas-bubbles in media containing grape-sugar. Now in the literature of this country the only test to which a given culture is subjected in order to exclude gas formation is by means of "shake cultures" in glucose-gelatine. This is not a sufficient test, for I could show a number of cultures (*e.g.* Gärtner's *Bacillus enteritidis*) which may not produce gas under these conditions, and

which are nevertheless no true typhoid bacilli. In order to exclude such bacilli it is necessary to test the cultures in glucose media, and expose them to the body temperature (liquid media in some form of fermentation tube, or shake cultures in media stiffened with agar); under these circumstances bacilli such as Gärtner's are definitely excluded, for they are able to form gas at 37° C., although they are unable to do so at 20° C. Of other media which are of importance in discriminating the typhoid bacillus I will mention the neutral litmus whey of Petruschky, and the litmus media of Capaldi and Proskauer. The former serves a double purpose: on the one hand it shows the amount of acid which the bacillus is able to produce in the presence of lactose; and on the other, since it is practically almost free from proteids, it demonstrates the scanty growth of the typhoid bacillus in such media.

In reference to the question of sero-diagnosis, I think we may lay it down absolutely that what is known as "Widal's test" is absolutely worthless. By this I mean the technique which was published by Widal some months after Gruber had brought the question forward at Wiesbaden. Widal's test, then, consists in investigating the action of the serum of typhoid fever patients when diluted only to the extent of 1 in 10 upon a typhoid culture; I think that it is quite proved that such low dilutions give no satisfactory evidence, unless negative. If, however, proper dilutions are made (Gruber's test)—the necessity for which was emphasised by Gruber himself in April, 1896 ('Verhandlungen des Congresses für innere Medicin,' 1896)—more satisfactory evidence of reaction is obtainable (the following dilutions form a useful series for practical clinical purposes, viz. 1 : 20, 1 : 50, 1 : 100, 1 : 200, 1 : 500, and 1 : 1000). In any case, unless a given serum reacts strongly in high dilution, the evidence given only amounts to a probability. It would appear that some observers have endeavoured to differentiate bacilli by means of their reaction towards sera obtained from typhoid fever patients. This is an extremely

lamentable departure from all that we know yet about the so-called "specific" actions of the sera of immunised animals, whether the trial is made in the test-tube or in the living animal. Now, if you wish to recognise a race of bacteria by means of its serum reaction, you must use a *very potent sample* of serum and *very little of it* in the animal test, and you must use *high dilutions* in the clumping and sedimenting test; so far as my experience goes, serum of sufficient potency is very rarely obtained from man. As an example of the degree of potency which it is possible to obtain from immunised animals, I may mention that I have a sample of serum which gives quite a good reaction when diluted 2,000,000-fold; such a serum will give a reaction visible to the naked eye in a few minutes when diluted 50,000-fold. For present purposes sera may be divided into three classes: (a) *weak*, which react up to 1 : 500; (b) *medium*, which react up to 1 : 50,000; and (c) *strong*, which react at 1 : 100,000 and upwards. For purposes of recognition, the first are almost if not quite useless; the second may be used, but it is only the third class which are of real service in giving as absolute evidence as is possible from the method. If observers would be a little more careful in making a series of dilutions (say up to 1 : 1000) when attempting to diagnose typhoid fever in man, and in using only highly potent sera highly diluted for the recognition of bacilli, there would be less valuable time wasted.

#### DR. CHILDS.

The progress of this discussion has served to illustrate the many points of attack and defence available to us against typhoid fever. My chief reason for speaking is that we have not given sufficient consideration to the protection of our water-supplies and the prevention of their pollution, although both Dr. Poore and Sir Richard Thorne have pointed to our public water-supplies as the

chief causes of our frequent typhoid epidemics ; whilst the latter has emphasised the fact that our high sick-rate and death-rate from typhoid fever are especially maintained by these epidemic outbursts. There are probably some present to-night who are sceptical about the convection of typhoid fever through water in certain cases in which it has been taken as proven : I admit I am sceptical myself ; yet I doubt whether there is anyone present who is not assured that at any rate some of these outbreaks are due to pollution of the water-supplies.

Now what are the means generally in vogue for the detection of this pollution ? I may divide them into two classes :—1st, the “*experimentum in vitro*,” *i. e.* by chemical and bacteriological analysis ; 2nd, the “*experimentum in vivo*,” *i. e.* by experiment on human beings. Both of these methods have the great disadvantage of coming too late ; that is to say, by the time the pollution has been detected most of the mischief has been done.

The “*experimentum in vitro*” has also the grave defect that it gives, in a large number of cases, a false sense of security.

The “*experimentum in vivo*,” by which we await the outbreak of the typhoid epidemic to prove the pollution of the water, is very general. This method is the more popular one, creates no friction, and, until the epidemic occurs, causes no expense to the ratepayers. Is it not certain that in this country at the present time there are numbers of water-supplies which are extremely liable to pollution, and that some of them sooner or later will give proof thereof by epidemics of fever ? The epidemic occurs, and then it is found out too late, after a great loss of life, labour-value, and trade, that the water is polluted. Too late also it is realised that the expenditure, which would have been necessary for the supervision of these water-supplies, would have been a very safe and wise investment. Next let us consider what legal powers there are for the prevention of this pollution. What legal obligation is there on the sanitary authorities to see

that their water-supply has no chance of pollution? What legal obligation on the water companies? The sanitary authorities themselves we find are curtailed and hindered in various ways,—as at Maidstone, for example. If the source of the water-supply happens not to be in that particular sanitary area, but in a neighbouring district, that sanitary authority has no power of access to the sources of its own water-supply, and if it visits them it visits them as a trespasser. Surely this is one of the most important and urgent points which require attention if we are to obtain effective legislation on this subject. Referring once more to Maidstone and Clifton, let me point out how, with the means at present available in this country, the sources of these epidemics were detected and cut off with the greatest possible alacrity and with the minimum amount of delay. Yet what was the result? Nearly all the mischief had been done before the source of the poison could be cut off.

I trust I shall have the assent of this Society when I suggest that one of the most urgent demands that we have for the prevention of enteric fever in this country is more effective legislation;—legislation by which it shall be enacted that the water companies shall be held responsible for the pollution of the water which they supply to their customers; that the local sanitary authorities shall have free access to the water-supplies from source to distribution; that there shall be an inspection by or under the Local Government Board, or by the County Council, to see that local jealousies or local interests are not interfering with proper inspection; that the arrangement and distribution of all water-supplies in the country shall be registered; that full plans, sections, and reports shall be made accessible to every water-rate payer and sanitary officer who is disposed to ascertain the chances of contamination.

Sir Richard Thorne pointed out that in the eighth decade the mortality from typhoid fever in England and Wales had considerably diminished, but that since that

time we had made but little progress. In other words, we have brought it down from 32 per 100,000 to 17, and there we have stuck. I have had an opportunity of watching the reduction of typhoid mortality in Munich for the last twenty years; in the eighth decade it was more than three times as great as our own; but at the present moment it is three times as little. We have reduced it rather less than one half; they in Munich have reduced it more than twentyfold.

If such happy results have been produced in a large German city, may we not hope for at least equal success in England and Wales?

#### DR. SEYMOUR TAYLOR.

I feel some diffidence in addressing such a meeting as is assembled here to discuss so important a question as is before us; but I would desire to draw attention, as a field of inquiry, to what I may term the clinical or practical physician's side of the question. We have heard much as to the bacillary invasion, or, as it has been aptly termed, the "*flora*" of typhoid fever; but as yet we have had little or no attention drawn to what I may call the *physical geography* and *geology* of the disease. And I apprehend that, as in agricultural discussions, the soil or ground on which a seed falls is almost if not quite as important a factor in the production of crops as is the seed itself. And it is to this aspect of the debate to which I would desire to confine my remarks.

It has been truly said that typhoid is endemic in this country, but it is also true that epidemics of more or less severity occur from time to time, and when the disease is epidemic the outbreak is as a rule local in character, and not at all widely spread. Why should this be? Unquestionably the specific poison of this disease is abroad in its due season, viz. autumn and early winter; and probably most of us receive into our systems, whether by



alimentary canal or by respiratory tract, a dose or doses of such poison. This must of necessity be so if it be accepted that typhoid bacilli may be given off to the atmosphere from the evacuations of a typhoid patient, or from the effluvia of a sewer. So that the specific bacilli, like the poor, are always with us. The same proposition obtains in regard to the bacillus of tubercle and of other specific diseases. Then naturally follows the inquiry, "Why do we not all suffer?" or at least, "Why is not the disease more widely spread?" My reply is, "Because we are not all of us good cultivation tubes;" the soil on which the parasitic sowing falls is not suitable for a good crop. In other words, the most of us are in good health, and we therefore fail to cultivate the plant.

I would therefore, with your permission, point to one or two factors which apparently determine the outbreak of typhoid in its epidemic form.

(1) Its relation to the existence of diarrhoea in a community, and its prevalence during hot weather.

(2) Its prevalence amongst the recent comers to an infected district, suggesting that the systems of old dwellers therein may become immune to the fever by constant exposure to mild doses of the poison.

(3) Its well-known antagonism to remittent and intermittent fevers, or at least its lesser prevalence where these fevers are rife.

As regards the first of these headings I would draw attention to the fact that people may drink polluted water for months, nay, perhaps years before a sudden epidemic of typhoid appears amongst them; and then it would appear that diarrhoea or some disturbance of the gastro-intestinal canal was the determining factor. I myself have drunk water from an infected spring with immunity, whilst my two companions, equally exposed to the danger, sickened of the fever.

In support of the second proposition I would instance the outbreak of an epidemic which I studied in the country, in newly erected and "jerry-built" houses in

which the soil-pipes were defective. The inmates of these houses had migrated from a different part of the country, and the fever was almost entirely confined to these people, notwithstanding that almost similar sanitary defects were discovered in the older residences, and the same sewer ventilated itself into the rooms of these houses.

In respect to the third proposition I would ask, "Why is it that enteric fever is comparatively rare amongst Oriental races, notwithstanding that the typhoid poison exists there?" I do not say that Orientals are not subject to the disease, but I do say, from study of the literature of the subject, that typhoid is not so prevalent amongst Eastern peoples as it is amongst the Westerns who reside in their midst. Nor is this partiality of a disease for certain races restricted to typhoid. We observe the plague decimating the natives of India, whilst Europeans apparently run only a slight danger.

Therefore we are forced to the conclusion that excremental poisoning is only one factor in the production of typhoid fever; indeed, pent-up sewage appears to be a more potent factor than contaminated water, else the Thames should be a fertile source of the disease.

In conclusion, therefore, I would ask those who are engaged in research to devote some of their energy to a study of the personal characteristics of communities, and to endeavour to ascertain if there be not some determining influence in the people which tends to originate an epidemic.

In the ætiology of any disease the predisposing causes are to my mind every bit as important as the exciting cause; and the soil is worthy of our attention every bit as much as the seed.

DR. GEORGE VIVIAN POORE (*in reply*).

In my reply I shall devote myself more particularly to one point which I brought forward, and which has been a good deal controverted, viz. the advisability or otherwise of putting excreta, including typhoid excreta, upon the soil. Now Sir Richard Thorne and Dr. Fletcher Little rather exaggerated anything I had to say on this. The former tried to put into my mouth words that I never uttered, words that I distinctly excluded. He said that I advocated that we should give up our water-closets in London and have recourse to surface wells. What I did say was that the best thing would be to burn typhoid excreta, but that in country places I did not see any harm in putting them upon the soil. Dr. Little suggested that I advocated putting typhoid excreta upon the gardens in Cavendish Square; but, needless to say, I did nothing of the kind. I did say, however, that excreta should be placed near the top of a well-tilled humus, and that the people should have an opportunity of seeing the result. There is great ignorance throughout the country as to how to treat excreta. Where the earth system and other dry systems are in use, the excreta are often treated in a way in which they cannot be destroyed. I find, in the army, rules that trenches are to be dug four feet deep, into which the excreta are to be cast and then filled up with earth and rammed down. That is like laying down wine for our heirs. Excreta cannot be destroyed at that depth, and when at some later period the earth is disturbed there is the poison ready to do its work. Much of the ill success which has attended the earth system in the East is due to its not being carried out properly, scientifically, and systematically. The Director-General of the Army Medical Department in his remarks showed that our troops suffered from typhoid fever enormously in the

tropics, and he said that this was the case in many places where the dry earth system was in vogue. That may be true, but we must remember that the European when he goes to the tropics suffers terribly from thirst, and will under stress drink at any puddle. Moreover it is a rule of religion with Orientals to carefully cleanse themselves after evacuations, for which purpose they do not hesitate to avail themselves of the water-tanks, so we must not judge the success of the dry methods by Indian experience. I gather from the army statistics that the typhoid rate in the Bermudas and in India was enormous; but one region stands out as an exception, viz. China. I find the case-rate is only 1·2 per thousand in China for the eight years 1886-93, as against 22·2 in Bengal. Now China is perhaps the one great country in the world where excreta are carefully collected and placed on the land. Now, sir, when Sir Richard Thorne says he agrees with me that typhoid excreta had better be burned, he advances a good way to meet me in my arguments. He goes so far as to say that there may be something in what I advance, but that the method being unfitted for London is fitted only for the days of Adam. Well, Adam lived for 930 years, but he lived at a time before any model bye-laws had been promulgated. But even though what I advocate is not applicable to London, that does not settle the question which I am contending for as a principle, viz. that if you wish to protect your water-supply it is safer to put your infective material on the top of the humus than to put it below. We have been told over and over again of the evils of putting it below, but I have not heard of a single instance of the spread of enteric fever from putting it on the top. I confine my remarks of course to this country; but I take it nothing better for tropical climates was ever devised than was devised by Moses, who said that every man should go outside the camp, and having a paddle on the end of his weapon, should lightly cover his excreta with soil. This is a protection against the conveyance of the infection by flies. What I advocate is this: the

daily removal of excreta, which are to be lightly covered on a well-tilled humus ; all offensiveness is gone at once, and in a few weeks they become unrecognisable as excreta. There is no getting away from dung in this world ; if you want to get rid of it you must put it to its proper use, and not bury it too deeply. There is, as I have said, a great deal of ignorance as to how excreta should be treated. I am of opinion that some of the money which county councils spend in technical education should be devoted to the education of the people in this matter. I go into towns where I see middens and closets which are filthy in the extreme, and inspectors of nuisances have very often no more idea of what ought to be done than the man in the moon. Their only idea is to get the excreta into the sewers and so get them out of sight. " Out of sight " and " trust to luck " are the principles of sewer sanitation.

We must accept the fact that the typhoid bacillus will live in earth. Martin's and Robertson's researches seem to show that it lives much more readily in a foul, polluted soil than in a clean soil. This foul, polluted soil in the big towns is provided for by leakage of drains, privies, and middens ; middens into which slops are thrown, thus becoming putrefaction chambers too horrible to think of, which sooner or later corrupt the soil. Well, how are we to cleanse the soil when it has become foul ? I believe there is only one way, and that is tillage. You must grow something upon it. Cover it with asphalte, cement, or what not, you merely bottle up the filth, and it will break out somewhere and somehow. If the soil gets polluted, and if the soil which is drenched with organic matter grows the typhoid bacillus, how about sewage farms ? In my paper you will find at least two instances, Beverley, and High Wycombe, Bucks, in which typhoid spread from sewage farms. In Buckinghamshire it was made pretty clear that the typhoid bacillus had leaked through the sewage farm with an enormous amount of water. Therefore the Local Government Board, in

demanding that all sewage schemes shall embrace intermittent downward filtration through land, tries to enforce a rather doubtful rule. Dr. Corfield disagrees with me in saying that the epidemicity of the disease is due to the water-closet. Of course that cannot be absolutely proved either way, but I was quoting William Farr. I have here a chart of the typhoid mortality in the counties to which he has referred. They are largely industrial counties, and it is true that in them there is most typhoid. Middlesbrough is a type of these places. Not only are the middens too foul to think of, but they are placed quite close to the back doors. The model bye-laws of the Local Government Board allow a privy door to be within six feet of the back door, which obviously exposes the kitchen to fæcal contamination. Now the counties which have least enteric fever are almost all rural counties, where the population is sparse, and where water-closets are quite the exception. The figures are given in the body of my paper, and will be found to bear out what I say. A description of Middlesbrough is also quoted. In the Scandinavian and Dutch cities, where dry methods are largely used, the enteric death-rate for the six years 1891-6 was only '09.

One other point as regards the earth. If the earth be the dangerous thing which some people would have us believe, it certainly is a remarkable fact that those who deal with and live upon the soil, agricultural labourers and gardeners, are the healthiest people in the country; they eat their food seated on the soil with hands not over-clean, yet they never seem to get any infectious disease. I know of no disease which is peculiar to them as a class except old age and rheumatism.

The principle for which I have been contending—that putrescible refuse should be placed upon and not beneath the great natural filter and protector of our water—is not without effect upon the towns, even though it may not be practicable within them. Much of our enteric fever is due to milk; and what form of sanitation, I would ask,

are we to recommend for the lone farmhouse whence our milk is derived ?

Is the farmer to be encouraged to persevere with his cesspool, which is sure to leak into his well, or is he to be taught to dispose of the excreta scientifically and systematically near the surface of a well-tilled humus ?

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LATENCY AND FREEDOM FROM RECURRENCE  
AFTER OPERATIONS FOR  
REPUTED CARCINOMA OF THE BREAST  
ILLUSTRATED BY 108 TABULATED CASES

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It is a matter of general belief that the results of removal of the breast for carcinoma in the first half of this century, and, indeed, up to quite modern times, were essentially bad. Such surgeons as Cline, Everard Home, and Munroe would hardly consent to its performance. Brodie limited the scope of the operation to a very few selected cases ('Works,' vol. iii, p. 256). He laid down conditions which well apply to the selection of favorable cases even in the present day. It was the same in other countries. Thus Halsted states that Agnew operated only for "moral effect," and Sands, in a long experience, declared he had not saved a single case ('Annals of Surgery,' 1894). The form of operation executed in the earlier years of this century was due to a kind of revulsion from the old and very complete, but barbarous method

of sweeping off the entire mamma, skin and all, and freely applying the cautery. It may truly be said that numbers of operations up to 1880 were markedly incomplete. In their anxiety to unite flaps, surgeons ignored the fact that they were actually shutting up cancerous foci in the wound, the growth being really never removed at all. This state of things was prominently brought forward in a remarkable paper by Moore, of the Middlesex Hospital, which hardly received that attention in this country which its importance merited ('Med.-Chir. Trans.,' vol. 1). Moore's paper was to show the influence of inadequate operations upon the return of cancer. The concluding paragraphs are worth referring to, as clearly showing that an English surgeon, as long ago as 1867, correctly estimated the method of spread of the disease, and actually described the "complete" operation now in vogue, which we have so largely adopted from foreign recommendations. "Centrifugal dispersion," writes Moore, "determines the spread of cancer." "The entire breast needs extirpation." Besides the skin, other adjacent structures need extirpation. Later on, Gross in America advocated the same doctrine, and it is right to point out that Mitchell Banks, about the year 1880, was one of the early "revivalists" of the complete operation in this country.

In his notable paper Moore refers to a case where recurrence took place in the scar nine years after the "inadequate" operation. He truly remarks that with such a result the operation could hardly be considered an incomplete one. Here is exemplified the curious exception to the general rule. Hardly any writer on cancer of the breast, hardly any surgeon of long experience, but is able to supply cases of this nature, where years of immunity from disease are observed even after operations now considered with reason to be "incomplete." Even Brodie, with his well-known unfavorable view of the operation, referred to undoubted cases well thirteen and six years after operation ('Works,' vol. iii, pp. 261, 262). Many such

cases are scattered about in literature, or in unpublished records of practice. Thus Greenhow referred to a case under his notice, where carcinoma occurred in a distant organ, eight years after a scirrhus tumour of the breast had been destroyed with caustics. In such a case the axillary glands would, of course, have been left untouched ('Path. Soc. Trans.,' vol. xxv, p. 366). Dr. Keser, of Harley Street, informs me that he closely followed the practice of Professor Socin of Bâle, and found five undoubted cases in that surgeon's practice well five to thirteen years after the original operation. Professor Socin operated by the old method, only opening the axilla where enlarged glands were detected.

It is not my object in the present paper to contrast the results of the complete and incomplete operations. I would only point out that to be logical the complete operation must aim at removing every particle of lymphoid tissue that may be infected. I would take as the type Halsted's operation, in which, as is well known, not only are the growth and skin freely removed, but also the pectoral muscles, fascia, and lymphoid tissue from the sheath of the vessels. The type of what is called the incomplete operation would be the removal of the whole breast and affected skin, but not of the contents of the axilla, unless the glands are felt to be enlarged, and not of necessity the extreme outlying prolongations of mammary tissue. It is right to point out that among modern operators difference of opinion still obtains in the comparative value of these two methods of operating. To quote only two instances. Treves, writing in 1891, states that he had given up the practice of clearing the axilla, as the results obtained had appeared to him in no way more satisfactory than those which followed the lesser operation ('Manual of Operative Surgery,' vol. ii, p. 758). On the other hand, Jacobson, writing in 1896, takes an opposite view, and supports extensive operating ('Operations of Surgery,' 3rd edit., p. 549). I might easily quote other instances of differences of opinion, but we all know that these are

manifest, and the matter need not be further demonstrated. When differences of opinion exist, there is always much to be said on either side. The trend of general surgical opinion seems to be rather in favour of early and extensive operating. No one who has studied the results of Dennis, Meyer and Halsted in America, can doubt that the "complete" operation lessens the liability to local recurrence in a most marked manner. This subject is so well treated in Cheyne's recent Lettsomian lectures at the Medical Society of London ('Trans. Med. Soc.,' 1896), that I need not refer to it further. There is one point, and that a very vital one, which I fear has hardly received the attention it deserves with regard to the result of the complete operation on cancer of the breast. How far does the most complete operation known influence the occurrence of metastases in the viscera, bones, or other parts? If we carefully consider Halsted's cases ('Annals of Surgery,' 1894) we are at once struck with two things. First, the cases are many of them of an exceedingly bad type; secondly, the results are reported with perfect sincerity. Out of fifty cases, in ten the ultimate result or cause of death is not known. In seven instances the cases are reported under twelve months, some in as short a time as three or four months after the operation. Such cases are, of course, valueless for computing the frequency of metastases. This leaves thirty-three cases for consideration, and out of these nine died of metastatic cancer. That is to say, that the most complete operator known, not picking his cases, has a mortality from metastases in nearly 27 per cent. This is taking also the most favorable view, that none of the cases where the cause of death is not stated died of cancer.

The present tables are compiled with the following objects:

(1) To show that the results of the so-called incomplete operations, are not so universally bad as is generally estimated.

(2) To illustrate what, for want of a better term, I may

call the vagaries of cancer cases, and the extraordinary periods of latency of the disease in many instances.

(3) To show that some of the cases, when life has been prolonged to an extraordinary period, have been associated with operations upon local recurrences.

I am especially anxious not to try and prove too much by the cases narrated. In the first place, it will be noted that in some of the cases microscopical proof was not forthcoming. This is notably so in Mr. Teale's cases. Now, with regard to this point, it may be said that, however doubtful the clinical diagnosis of carcinoma may sometimes be, there is little difficulty about the naked-eye appearances of a typical specimen, when it is cut across. In cases of doubt, as in the so-called "indurated patches," I have usually found that there is always as much hesitation and difficulty in the minds of microscopical observers, as in those who have made the coarser inspection. I have taken special pains to verify the cases by queries. It will be seen that the authors state that the cases were undoubted. They speak of such phenomena as implication of skin and glands. The appearance "was typical when cut across." Indeed, Mr. Teale stated to me that his cases were so evident that microscopical proof was hardly needful. I have rejected some cases that have been communicated to me, where the growth contained cysts, or was soft and brain-like. This has been done with the idea of eliminating as far as possible cases of duct papilloma, and recurrent sarcomatous disease. I am ready to allow that it is possible that some of the cases narrated may have been recurrent sarcoma. Here, the comparative rarity of this disease must be reckoned with, and the cases lose little of their interest if an occasional error, like the inclusion of a sarcomatous growth, has crept in. To assume that these cases are untrustworthy, would be to assume that many surgeons of experience are unable to recognise the ordinary hard carcinoma of the breast by clinical signs and naked-eye anatomy. I think I have reason to claim, therefore, that the evidence of these cases being genuine

is sufficiently strong to render them of value, and to press them upon the attention of the profession as being of the utmost interest and importance.

It may be said that the instances are few in number. I would reply that they are rather selected for illustration than for number. For this reason I have taken them from writings, and from the experience of American surgeons, to show how wide-spread these cases really are. I have been able to obtain the experience of only a few senior surgeons who were personally known to me. The names of many, who doubtless could add to the number of these remarkable cases, are absent from the list. It is striking, too, that the majority of instances are culled from private practice, in which alone, generally speaking, the ultimate results of operations for carcinoma are known. In hospital cases the recurrent cases show themselves, the patients who remain free do not. It is right to assume that if a given surgeon can collect favorable cases from his private practice, he would be likely to collate a still larger number from the practice of hospitals, where the majority of his operations have been performed. As regards cases where periods of three, four, and six years have elapsed, I believe a very considerable number could easily be collated. I have four in my own practice, and many have been sent to me from all parts of the country, which I did not consider striking enough to be related in the tables. With reference to this, the experience of Sir James Paget may well be quoted. His pessimistic view of the recurrence of cancer of the breast is too well known to repeat here. Kindly writing to me in 1896, he says: "From my recollections I should say that survivals of three years, such as are now termed 'cures,' were not very rare. I can remember very few who survived without recurrence more than five years. Of those who survived more than ten years there were only two or three known to me."

In Group I will be found forty-four cases where no recurrence took place after the primary operation for

periods varying from six to twenty years. I use the term recurrence in its widest sense. Doubtless many surgeons would prefer to call some of these cases fresh outbreaks of disease. In comparison with the numbers in recurrent cases (Group II) these cases are in the minority. Some of the operations are markedly incomplete. Thus, in Mr. Nixon's case (No. 3) a cancerous tumour was excised from the mamma, the gland being left behind, as well as enlarged axillary glands. When the patient died seven years later cancer was not present in any of her tissues and organs.

In periods of time the cases in Group I may thus be presented :

Surviving and free from recurrence—

From 6 to 12 years	.	.	23 cases.
„ 12 to 20 „	.	.	16 „
20 years and over	.	.	5 „

Group II. It may be pointed out that the manifestation of recurrences is a strong proof of the malignant nature of the primary tumour, and generally speaking of their carcinomatous character. It will be at once noted that recurrences or fresh outbreaks of disease, if the term is preferred, took place at the most remote intervals after the operation. It is remarkable how comparatively seldom the second breast was affected. In this organ above all others, fresh outbreaks of disease might naturally be expected to occur. In 49 cases where late recurrences or fresh manifestations were observed, the following periods are exemplified :

Recurrence after operation was observed—

From the 4th to the 8th year	in 19 cases.		
In the 9th year	.	.	8 „
„ 10th „	.	.	10 „
„ 11th „	.	.	2 „
„ 12th „	.	.	1 case.
„ 13th „	.	.	1 „
„ 16th „	.	.	1 „
„ 17th „	.	.	1 „

In the 19th year	.	.	1 case.
„ 22nd „	.	.	2 cases.
„ 23rd „	.	.	1 case.
„ 25th „	.	.	2 cases.

In the 15 remaining cases recurrences of a local nature in the scar or glands took place early, say within two years, and were removed with striking results.

The removals were in some cases repeated. Extraordinary periods of prolongation of life are observed in some of these cases. Attention may especially be called to Case 12, in which there have been twelve operations for local recurrences during twenty years; to Case 50, where after six operations for recurrences the lady enjoyed good health for forty-nine years; and to Case 8, where three early operations for local recurrence were followed by twenty years' immunity.

Of 23 cases, in which recurrences had been removed, sometimes repeatedly, we find—

4 cases remained well for	5—10 years.
11 „ „	10—15 „
3 „ „	15—20 „
4 „ „	20—30 „
1 (an extreme case) „	49 years.

In considering the suggestions offered to our minds by the collation of these cases, I wish to carefully avoid the realm of speculation, already sufficiently encroached upon in reference to cancer cases.

One thing is clear. There must be something very remarkable about cases of this nature, which though exceptional, may at any time occur. It seems to me that one possible factor to consider is the question of rate of growth of the tumour. Unfortunately, it is impossible to obtain from patients an accurate estimate of the length of time a given tumour has existed. But as an illustration of the fact that tumours of slow growth are favorable for excision, notwithstanding their extent, a case quoted by Mr. Hutchinson from the practice of Paget, of Leicester, may well be here related ('Archives of Surgery,' vol. v,



p. 47). The patient was the wife of a surgeon who had contracted a cancer of the breast for several years, until it attained to such a size and state, together with an enlarged axillary gland, that the operator looked upon the operation as the "boldest step" he ever achieved. An extensive excision was made, and the patient remained well for no less a period than twelve years. Death occurred after this long period from cancerous nodules in the skin and pleura on the affected side. When long immunity is observed after operations for rapidly growing tumours, I believe such are generally encapsuled, and frequently either "villous duct cancers or sarcomata."

The second factor at work in these cases, may be something in the body of the host inimical to the rapid growth of the malady. Perhaps in the last twenty years we have studied too much the growth, and too little the soil in which it grows. We are completely ignorant of the conditions which lead to retrogression and atrophy of carcinoma of the breast, and a collective investigation and study of these cases in all their bearings as to bodily health, other maladies, hereditary taints, and so on, is a matter not beneath the notice of the leading London societies. I cannot but believe and assert that, looking at the results of operations really incomplete, the years of immunity sometimes observed are due either to peculiarities of the growth, or to unsuitableness of tissue for its rapid propagation. This more than the operation has to do with the favorable results. I see no reason why the general rule of operations for cancer is not easily carried out in the mamma—operate early, operate widely; but as regards the term "cure," as adopted by Volkmann and his disciples, when freedom from local recurrence is observed for three years, I think we ought to employ it with the extremes of caution, and I think many of the cases I have tabulated bear out and forcibly illustrate this opinion. It is worthy of remark that Jacobson, in a careful review of the subject, comes to much the same conclusion, and states his opinion that at least six years

should elapse after the primary operation before the term "cure" can be used ('Operations of Surgery,' 3rd edit., p. 553).

It seems only too probable, that once a cancer forms in the mamma, the blood and lymph which percolate through it carry the infective elements broadcast far sooner than we dream of. The obvious manifestations in the adjacent lymphatics must not be taken as the only dissemination which occurs. This is only a proof of the smallness of our powers of observation even with the microscope. Outbreaks of cancer in the glands, as seen years after, can only mean latency of the infective elements. Outbreaks of cancer in the viscera may mean fresh disease; yet it is curious, if such be the case, why we find the cancer attacking such parts as the bones of the vertebral column, the pleura and lung, or the brain, where primary sphe-roidal-celled carcinoma is almost unknown, unless the breasts have been affected.

In conclusion I express the hope that this paper may afford encouragement to surgeons in operating upon carcinoma of the breast, and may also serve as a useful reference in persuading patients to have the operation performed. The many suggestive points which such cases bring out, afford the strongest possible argument for the careful study and collection of similar instances, by all members of the profession.

TABLES OF CASES REFERRED TO IN  
THE PAPER

*The Cases referred to in the Paper.*

## GROUP I.

*Cases where the mamma has been removed, presumably for carcinoma, the patient living long without recurrence, or dying of other affections many years after.*

No.	Primary operation.	Remarks.	Results.	Source or authority.
1	Breast removed for undoubted carcinoma in 1888; axilla not cleared	No microscopical examination made, for evidence on naked-eye section complete	Patient quite well 6 years after. ( <i>Note</i> .—Of these cases, 5, 6, 7 years, 8 have been collected. I insert this as an example)	Dr. Sandberg, of Brixton.
2	Breast removed for carcinoma in 1885	No microscopical evidence; nature of growth stated to be quite clear	Patient quite well 9 years after	Mr. Travers, of St. Leonards.
3	A woman aged 50, of powerful physique and with enormous mammae, a noted street fighter. A carcinomatous tumour formed after a blow, and a mass of hard glands formed in the axilla. By the advice of Colles, of Dublin, only the tumour was removed, the glands and the remainder of the large mamma being left. The operation was therefore very incomplete	Naked-eye and microscopical evidence of glands and tumour	This patient remained well, and subsequently died 7 years after the original operation from cirrhosis of the liver. The body and mammae were examined; there was no trace of cancerous disease	Mr. Fred. Nixon, Dublin.
4	In a patient 53 years of age a carcinomatous tumour the size of a lemon was removed from the right breast; the axillary fascia and glands were also removed	Carcinoma stated to be undoubted; examined by microscope	Well without recurrence 9 years after. At the same meeting Coley stated that he had under his care a woman, 71 years old, whose breast had been removed for carcinoma by Hodges, of Boston, nine years before	Murray, Proc. of New York Surg. Soc., April 24th, 1894.
5	Breast removed for a mass of cancer the size of a walnut from a lady aged 41. This was in 1873. The axillary glands were not removed	Stress laid on the undoubted nature of the growth	Seen alive and well 21 years after the operation	Dr. Allan Jamieson, of Edinburgh.

6	Removal of the right breast only for malignant disease	The tumour had the usual characteristic symptoms	18 years after she was in perfect health	Dr. Morton, of Philadelphia, <i>Annals of Surgery</i> , 1894, p. 94. Dr. C. Bradford, Acocks Green, Worcester. Sir George Humphry.
7	A lady had both breasts removed for undoubted carcinoma by Mr. Furneaux Jordan	No microscopical proof; case stated to be "very obvious"	Lady in perfect health 15 years after	
8	Two patients, aged 60 and 55 respectively, were operated upon for carcinoma of the breasts. The skin was freely removed, and the wound encouraged to suppurate; the glands were not taken away	—	These were well without recurrence 5 and six years after respectively	
9	A case of removal of the breast for "scirrhous" when the patient was well 11 years after	—	11 years	
10	In June, 1874, the late Mr. Durham removed the mamma of a lady for undoubted carcinoma; the axilla was not opened	The tumour was "cut" afterwards, and was certainly hard carcinoma	22 years after this lady was alive and well. ( <i>Note.</i> —Mr. Durham had lost sight of this case)	C. Heath, <i>Med. Press and Circ.</i> , Jan., 1895. Mr. Wallis, of Cambridge.
11	Breast removed; axillary glands cleared out	Nature of disease proved by microscope	Well with no recurrence 10 years after	Mr. H. Morris.
12	Breast removed for undoubted carcinoma	—	This lady was operated upon for "gall-bladder trouble" in 1896, 7 years after the operation in the mamma; there was no trace of return	"
13	In June, 1878, a lady had the breast removed for cancer	—	She was well and free from recurrence twelve years after	"
14	A lady aged 68 had the breast removed for cancer by Mr. Bryant. No mention of clearance of axilla.	The statement of the author that the disease was cancerous	She died of "gall-stones," as verified P.M. There was no trace of recurrence of cancer; this was 15 years after	Mr. T. Bryant.
15	A lady had the breast removed for cancer at the age of 60 by Mr. Bryant	—	She died of "old age" at the age of 80, 20 years after	"
16	Right breast and some axillary glands removed from a lady, aged 47, in 1886	No microscopical proof. The case is stated to be undoubted	She was quite well 8 years after	Mr. Barwell.

No.	Primary operation.	Remarks.	Results.	Source or authority.
17	Breast removed from a hospital patient. The skin was involved, but not the axilla	This was a case of cancer, where there was extensive involvement of the skin	She was quite well 11 years after	Mr. Barwell.
18	In March, 1885, the whole breast was removed from a patient 68 years of age "for a hard cancerous tumour."	On inquiry it is stated that the skin was involved, and the nature of the growth on naked-eye section clear	This patient was well 10 years after	Dr. Trotter, Cranlington, Northumberland.
19	In January, 1888, the breast and some enlarged axillary glands were removed by Mr. Butin	Sir James Paget confirmed the diagnosis, and speedy recurrence was prophesied	This patient was well in 1895, 7 years after	Mr. Tapson, of Clapham.
20	A widow, aged 74, had the whole breast removed for extensive "scirrhous" involving the skin by Walker, of Peterborough. The edges of the wound could not be brought together	Case seems quite clear; the implication of the skin with nodules, &c., spoken of	10 years after the patient was quite well	Dr. Hubbersty, of Sunderland.
21	A married woman, aged 60, had the breast removed for "ulcerating scirrhous" by Walker, of Peterborough, in October, 1879. No mention of axillary glands being removed	Case undoubted	She died 17 years after the operation. The actual cause of death was not ascertained, but there was no trace of disease in the scar or elsewhere	"
22	A patient, aged 65, had the breast removed for cancer in April, 1883. The axilla was not cleared	—	She was quite well 6 years after, and died of old age	Sir T. Smith's cases, Lancet, Aug. 8th, 1896.
23	Breast removed from a patient, aged 50, in January, 1883. The axilla was not cleared	—	13 years after she was quite well with no return	Sir T. Smith.
24	Breast removed from a patient, aged 45, in May, 1877. Axilla not cleared	—	Well without recurrence 17 years after	"
25	Breast removed from a patient, aged 65, in August, 1880. Axilla not opened	Case of atrophic scirrhous	Died of "influenza" 14 years after. No sign of return of disease	"

26	Breast removed from a patient, aged 47, in February, 1889. The axilla was opened and some enlarged glands removed	Case of atrophic cancer	Alive and well 7 years after operation	"
27	Left mamma removed in 1886 for carcinoma; enlarged axillary glands removed also. Mr. Mayo Robson was the operator	Microscopical and naked-eye proof	10 years after this patient was alive and quite well	Dr. Carr, Kippax, Yorks.
28	Breast removed from a lady aged 49. There were some glands to be felt in the axilla, which were not enlarged. Date of operation June, 1863	The microscope was not used, but I have the assurance of the author that the naked-eye appearances were perfectly clear	13 years afterwards this patient was quite well	Mr. Pridgin Teale
29	A single lady, aged 69, had the whole mamma removed for ulcerating carcinoma in May, 1877. Three cancerous glands removed from the axilla	Case, according to author, a very marked and extensive one of carcinoma	18 years after she died of "senile decay" with no recurrence	"
30	In September, 1888, breast removed for undoubted carcinoma. Age of patient not given	Microscopical verification by Dr. L. Thomas, of Cardiff	Patient quite well 8 years after	Dr. Edgar Jones, of Cardiff.
31	Delagarde, of Exeter, states that he knew a patient survive 27 years after removal of the breast for cancer	Statement of author. The term "thoroughly scirrhous" is used, and the patient died insane	The 27 years; no recurrence	St. Bart.'s Hosp. Rep., vol. iii, p. 319.
32	A lady, aged 63, skin implicated. Breast and skin removed, but not axillary glands, in 1874	Naked-eye appearances on section those of "scirrhous" cancer	There was no return in 1897, a period of 23 years; meanwhile one of her sisters died of cancer of the breast, another of cancer of the uterus, and the mother died of cancer also	Mr. John Croft <sup>2</sup>
33	A lady, aged 48, operated upon in 1877. Skin much involved; axillary glands involved also. The glands were removed	Growth and glands carcinomatous to the naked eye	Known to be well in 1888, 10 years after operation; case not further traced	"

<sup>1</sup> It may be concluded generally that in all these cases the extensive complete operation, as now practised, was not done. If enlarged glands were felt in the axilla these were cleared away. In several of Mr. Morris's cases, however, the axilla is stated to have been cleared out. Mr. Smith's cases are typical of the "partial operation."

<sup>2</sup> Note on Mr. Croft's cases.—None of these cases were microscopically examined. Their value lies in their source, and the fact of the typical and naked-eye appearances after removal. We can hardly doubt that they were genuine cases of carcinoma.

No.	Primary operation.	Remarks.	Results.	Source or authority.
34	A lady, aged 56, operated upon in 1878. No history of axillary glands being removed	There was a cyst in the breast as well as "scirrhous" cancer	She was well in 1891, a period of 13 years after the operation. Her mother died of cancer, and her sister of cancer of the breast.	Mr. John Croft
35	A lady, aged 31, operated upon in March, 1879. The tumour was small, but the whole breast was removed. The axillary glands were left alone	The naked-eye appearances were those of scirrhous	No return in 1896, a period of 17 years. A number of the relatives had died of cancer meanwhile	"
36	A single lady, aged 65, operated upon in 1881. No glands removed	Scirrhous of the sternum; border of the mamma. Adherent skin implicated	No return in 1897, a period of 16 years.	"
37	Lady, aged 35, operated upon in 1883. Glands affected markedly and removed	Growth was soft; carcinoma (tuberous cancer)	No return in 1897, a period of 14 years	"
38	A lady, aged 41, operated upon in 1889. The glands were not affected or removed	A "scirrhous" tumour the size of a half orange; naked-eye appearances typical	Well in 1897, a period of 14 years after	"
39	A lady, aged 41, operated upon in 1885	"A small scirrhous"	"2 years ago," in 1895, was well and "free from cancer," a period of 10 years	"
40	A lady, age not given, operated upon in 1885. Glands not affected or removed	Naked-eye appearances	Well in 1897, a period of 12 years	"
41	Miss F—, aged 47, Dec. 5th, 1883. Removal of breast; axilla not opened	Well-marked scirrhous carcinoma involving about half breast confirmed by microscope	Remains well, Feb. 3rd, 1897, a period of 14 years	Mr. W. Haward
42	Amputation of left breast only in a lady in November, 1875	A large tumour; the skin over it is involved; of the acute type. Microscopical evidence of carcinoma also	Known to be well in 1890, a period of 15 years	Late G. C. Rigdon, of Lewes. Mr. A. Willett.



43	Right breast removed from a lady in April, 1891. Axilla not opened	Microscopical evidence: a large, hard, rounded tumour; bloody fluid from nipple; case appears to incline towards the atrophic section "scirrhous"	Complete freedom in 1897, a period of 6 years	Sir James Paget; Mr. Jowers, of Brighton; Mr. A. Willett.
44	Breast removed from a lady, aged 57, on May 23rd, 1869. Glands also removed from axilla. The tumour was situated at the edge of the left breast	Microscopical "scirrhous"	Complete freedom in 1897, a period of 8 years	Dr. Lediard, of Carlisle; Dr. Mitchell, of Cockermouth.

## GROUP II.

*Cases of long life after removal of the breast for carcinoma, though recurrences took place, in some instances many years after the original operation. The value of repeated operations on recurrences is in some of the cases strikingly shown.*

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
1	A lady had the right breast removed for carcinoma by Mr. Jones, of Manchester, in 1874	Disease stated to be cancerous without doubt	17 years after the operation recurrence took place in the scar in the form of nodules. The abdomen swelled, and she died with ascites and carcinoma of the peritoneum	Mr. Banting, of Torquay.
2	Mr. Haward removed the left mamma from a woman at St. George's Hospital in 1887; the growth was carcinoma. Enlarged glands were removed from the axilla	Case verified by reference to notes. Previously to this the same surgeon had removed an ovarian cyst from the patient, but there is no note as to its nature	9 years after the mammary operation, the patient having remained perfectly well, tubers of carcinoma appeared in the scar, in the opposite mamma, and in the root of the neck	Author. This patient came to my out-patient department in 1896.

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
3	A lady, aged 38, had the breast removed by Pollock, of London	Statement that the disease was undoubtedly carcinoma	6 months afterwards a recurrence the size of an egg took place in the scar. This was undoubtedly cancerous, and was removed by Wyeth, of New York. 14 years after this last operation she was in perfect health	Wyeth, Proc. of New York Surg. Soc. March, 1894.
4	A case related at same meeting of removal of extensive and undoubted carcinoma of the breast	—	10 years after, recurrence in scar. Removal. 14 years after primary operation, in good health with no sign of return	Stinson, Proc. of New York Surg. Soc. Mar. 14th, 1894. Prof. C. Black, Glasgow.
5	Breast removed for undoubted carcinoma from a lady between 50 and 60 years of age. Date Sept., 1884	Nature of primary growth stated to be quite undoubted	6 years after, recurrence took place in the scar and was again operated upon. In 1896 another recurrence took place, and was again operated upon (a portion of the second recurrence was sent to me for examination. It was fibrous in structure, and might have been either keloid or atrophic carcinoma.)	
6	A lady, aged 57, had the right breast and axillary glands removed by Mr. H. Morris in 1885	Case quite undoubted	2 years afterwards, a recurrence took place in the scar. This was again removed. She was quite well in 1895, 10 years after the original operation	Dr. Lendon, of Norland Place, London. "
7	A single lady had the right breast removed by a very extensive operation by Mr. Pye in 1885	Not examined with the microscope. Disease extensive and undoubted	10 years afterwards, the patient in the interval remaining quite well, Mr. Pearce Gould removed a recurrent mass from the pectoral region. This was carcinoma. Patient after this remained well	
8	In May, 1876, a woman of 50 had the breast and some enlarged axillary glands removed for extensive carcinoma. This had existed for 17 months, and had been treated by injections of acetic acid	The growth was very extensive and clearly carcinomatous	Local recurrence took place in 1877, in 1878, and in 1879. The nodules were in the region of the scar, and were always freely removed. In 1896, 20 years after the first operation, this patient was alive and well	Mr. Edmund Owen.

9	A widow, aged 43, had the right breast removed for scirrhous in 1884	Microscopical proof	In 1885 the disease appeared in the opposite mamma, which was also removed, and the true nature of the growth again proved by the microscope. In 1896, 10 years after second operation, 12 after the primary operation, this patient was alive and well	Dr. Hare, of London; the late Mr. Durham; Mr. Howard Marsh.
10	Breast removed for carcinoma in 1874	Statement of author	Recurrence in scar a few months after. This was freely removed. 14 years after this lady was well and free from disease	Mr. Nelson Dobson, Bristol Med.-Chir. Rev., 1889, 240.
11	In connection with this case two others are mentioned, of 6 and 7 years immunity from disease respectively	—	One of these died of cancer of the tongue, and another had "a sarcoma of the soft palate" removed 2 years after the first operation	"
12	A lady had the breast removed for undoubted cancer. The skin was removed very freely. Some enlarged glands were also removed from the axilla	Sir George states, as the result of his experience, that the disease returns in the scar rather than in the glands. He was not sure of the wisdom of removing the glands unless infected. Very free local removal is indicated, and during the child-bearing period	Local recurrence took place "soon." This was removed, and she was well for a period of 10 years. Again recurrence took place in the scar. This was again removed. 2 years later recurrence in the femur and pelvis, which proved fatal	The late Sir George Humphry.
12	A lady had a cancerous tumour excised by Birkett from the upper margin of the right mamma in 1878. The whole breast was not removed	The primary growth was not examined with the microscope, but the subsequent ones proved to be carcinomatous	The age is important, and so is the rate of growth of the cancer, being worse in the young and during the child-bearing period	Dr. Hare, of London; the late Mr. Durham; Mr. Howard Marsh.
			She remained well for 10 years; then the growth returned, and the whole breast was removed by the late Mr. Durham. Recurrence took place in the scar or about it every year until 1894, when two recurrences were operated upon in the same year. In 1895 Mr. Marsh operated, and in 1896 an eminent provincial surgeon. This lady has had at least 12 local operations for recurrence, and is now, 20 years after first operation, in good health	

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
13	A woman of 70 was admitted into St. George's Hospital in May, 1894, when the right breast was removed for extensive carcinoma	Microscopical proof	12 years before this the left breast had been amputated for carcinoma (?) at King's College Hospital. The patient gave a very clear history of the original disease, and how she was told it was "cancerous" by Ferguson	Author.
14	A case of undoubted carcinoma in a lady aged 61, for which the breast was removed by Mr. T. Pick in 1886. The tumour was the size of a small orange, and had been 2 years in growing. The axillary glands were not removed	Clinical and microscopical proof	Patient remained well for 6 years; then a recurrent nodule the size of a filbert formed in the scar. This was removed. In 1897, 11 years after the second operation, this patient remains well	Mr. T. Pick; Dr. Morgan, of Torquay; author.
15	Observation at the Middlesex Hospital in March, 1896	Statement of Mr. Morris	A patient was seen with recurrent carcinoma in the scar of an amputation of the breast done for cancer 22 years before	Mr. H. Morris.
16	Right breast and axillary glands removed for carcinoma in September, 1862	Case seems genuine and undoubted	16 years after the operation there was a "limited return" at the upper border of the scar. This was apparently removed. 31 years after the first operation the patient was alive, but the arm was swollen, and there were "flat vivid plaques" of cancer in the old scar. 33 years after operation patient able to work as a charwoman	Nunn, Clin. Soc. Lond., Feb. 14th, 1896.
17	About the year 1882 or 1883 Dr. Iles, of Stratford, amputated the left breast of a lady for "very rapidly growing carcinoma"	Special stress is laid on the examination of the specimen with naked eye and microscope	2 years after, disease appeared in the opposite breast, the scar on the left side remaining healthy. She would not have the right mamma removed, but took the carbonate of calcium then recommended by the late Dr. Peter Hood. In 1896, 14 years after the first operation, she has no further	Dr. Stradling, of Watford, Lancet, 1897, and private letters

18	Breast removed for extensive carcinoma from a lady aged 68	Statement of author	<p>manifestation of disease. She has had several attacks of illness, but is now in fair health. The right breast is shrunken and "withered up." (I regard this as an instance of atrophic carcinoma.—A.M.S.)</p> <p>She remained perfectly well for 6 years. In the 7th year she died of cancer of the liver (verified), the scar and adjacent parts being quite healthy</p>	Mr. C. Brooke, Surgeon to Lincoln County Hospital.
19	A laundress, aged 59, was an inmate of St. George's Hospital in 1872, when the left breast and axilla were "cleared" for carcinoma by Mr. H. Lee (probably only enlarged axillary glands were removed)	<p>Nature of disease clear.</p> <p>The carcinoma had ulcerated extensively</p>	<p>The patient remained well for 5 years, when a small recurrence was removed from the scar. In 1886, 14 years after the first operation, she was again admitted with a small recurrence at the sternal end of the scar, which was removed. The axilla remained free. The ultimate result is not known</p>	Author.
20	In the year 1886 the breast was removed from a lady, aged 60, for "extensive scirrhus cancer"	<p>"The nature of the case was quite clear"</p>	<p>She remained well for 3 years. In the 4th year she became anæmic, and developed a dilated stomach and a pyloric tumour, of which she died. "I have no doubt that the cause of death was carcinoma of the pylorus"</p>	Dr. Freer, of Stourbridge.
21	Mr. Jonathan Couch, Surgeon, of Looe, in Cornwall, was present when his "master," Mr. Rice, removed the breast of a woman for "scirrhus." Mr. Rice especially dwelt upon the marked features of the disease. The skin was much affected, and the cancer was said to be of the stony variety	<p>Mr. Couch writes "that the facts of this case may be implicitly relied upon." In view of the pathology of ancient days this case may have been sarcoma. The nature of the recurrence seems clear</p>	<p>Nearly 25 years after, when the operator had "passed away" and Mr. Couch had succeeded to the practice, this woman presented herself with cancer in the cicatrix of the old wound. This was again operated upon, but it speedily returned and proved fatal</p>	Mr. John Couch, of Penzance.

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
22	In the year 1847 a woman, aged 43, had the left breast and axillary glands removed for scirrhus	The microscope was used. Nature of case quite clear	Recurrences were removed from the scar in 1848 and in 1856. In 1867, 20 years from the original operation, she had grown into a "hardy, fresh-looking old woman." The scar and axilla were quite sound. There were two fatty tumours near the scar	Delagarde, of Exeter, St. Bart.'s Hosp. Rep., vol. iii, p. 319.
23	The same surgeon states that he knew a patient die of cancer of the liver 19 years after removal of a scirrhus of the breast	Statement of author	19 years after operation recurrence in liver	Ibid., p. 324, footnote.
24	A woman, aged 50, had the right breast removed for scirrhus as large as a walnut	"It presented the true scirrhus structure"	9 years after, the scar remaining well, she died of cancer of the uterus. "After removal of cancer of the breast I have only in one instance seen it seize upon the womb"	Ibid., pp. 320, 326.
25	In the year 1892 the case of a woman, aged 58, was observed who had had the left breast removed by Mr. Morris 10 years before	(Mr. Murray, then Registrar at the Middlesex Hospital, kindly verified the diagnosis of the growth from the Hospital records for the author)	Her health was good for 10 years, and in the 11th year after the operation the growth returned in the axilla and left lung, and she died from cancerous pleurisy	Mr. Yearsley, 10, Bentinck Street, London.
26	Breast removed for carcinoma from a lady, aged 65, in 1883. Axilla not opened	Statement of author	Recurrence in scar in 1888 and 1891; both removed. Well in 1896, 13 years after first operation	Sir T. Smith's cases, Lancet, Aug. 8th, 1896.
27	Breast removed for carcinoma in November, 1887. Axilla not opened	"	Recurrence in scar removed in 1889. Well in May, 1896, 9 years after first operation	"
28	Patient aged 44. Breast removed for carcinoma in June, 1882. Axilla not opened	"	An enlarged axillary gland removed in February, 1883. Well with no recurrence in 1896, a period of 14 years	"
29	Right breast removed for carcinoma from a lady, aged 38, in October, 1882. Axilla not opened	"	She remained well for nearly 8 years, when recurrence took place in the scar, and the left breast became a <sup>second</sup> growth	"

30	Breast removed for carcinoma from a patient, aged 50, in March, 1879	"	She remained well for 13 years, and in June, 1892, died of "secondary growths" in the mediastinum	"
31	Left mammary gland removed for carcinoma from a lady, aged 55, in June, 1884. Axilla not opened	The tumour was the size of a pigeon's egg, hard with adherent and dimpled skin on section; it presented all the characters of "hard cancer"	She remained quite well for 7 years, then developed malignant disease of the peritoneum and abdominal glands with rapid wasting, from which she died	Dr. Alderton, of Hammersmith.
32	Right breast removed for carcinoma by Sir Joseph Lister. The axilla was cleared	No details of case except statement of patient that the surgeons assured her of the cancerous nature of the tumour, and evidence of extensive clearance of axilla	She remained well for 10 years, and then developed abdominal disease. Mr. K. Thornton operated, and found carcinoma of the right tube and broad ligament	Dr. Brigham, of Sydenham.
33	A woman had the left breast removed for undoubted hard carcinoma at St. George's Hospital in 1889. The right breast was absent, the scar being prolonged into the axilla	Microscope proof also	10 years before the left breast had been removed by Mr. Cooper Forster at Guy's. She had been perfectly well for over 9 years before the disease was noted in the other breast. Mr. Smith, the Surgical Registrar, looked up the notes and verified the nature of the disease in first operation at Guy's	St. George's case-books for 1889.
34	Breast removed from a lady on May 24th, 1869. The axilla was not cleared	Statement of operator that disease was certainly cancerous	In 1870 a recurrent nodule was removed from the axillary side of the scar. 26 years after the first operation this patient is perfectly well	Mr. Teale.
35	Breast removed from a lady, aged 41, in October, 1868. Axilla not opened	A bad case. Much skin and some of the pectoral muscle removed. Axilla not cleared, but one or two enlarged glands removed	In 1873, 3 years after operation, she was well and strong, with no recurrence. In 5 years after first operating the glands of the neck began to enlarge, and she gradually succumbed to the disease	"

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
36	Breast removed from a lady, aged 42, in October, 1870. Axilla not opened	Very marked carcinoma	She remained well for 10 years, then a recurrent nodule formed, and was excised from the scar. 15 years after the first operation she was well and strong	Mr. Teale.
37	Breast removed for carcinoma from a lady, aged 54, in August, 1874. Tumour situated towards sternal aspect. The axilla not implicated or opened	Marked carcinoma	3 years after the operation the disease appeared in the axillary glands, which were removed. She remained well for a further period of 4 years, then she died of recurrence in the neck	"
38	Breast removed from a lady, whose age is not given, in January, 1880. The axilla was not opened	"	Remained well for 9 years, then operator had a report she had gone to London on account of return of cancer in the glands	"
39	Breast removed from a lady, age not given, in November, 1894. Six or eight affected axillary glands were removed	The scirrhus was the size of a walnut, and situated near the axillary margin of the mamma	Remained well until 1889, when, 5 years after operation, recurrence took place in the scar	"
40	Breast removed from a lady, aged 71, in June, 1886. An enlarged gland removed from the axilla	—	She died free from cancer in 1895, 9 years after operation	"
41	Breast removed from a lady, whose age is not given, in 1885. No glands removed	The scirrhus was the size of a walnut, and a large area of skin was excised	She remained well until 1889, when, 4 years after the first operation, a small nodule "near, not in," the scar was excised. It implicated the pectoral, and was excised together with some implicated axillary glands. In November, 1896, this patient was living, 11 years after the operation. She had then a small nodule attached to the rib	"
42	A woman, aged 56, had the right breast removed at St. George's Hospital for undoubted scirrhus in 1888. The axilla was also cleared out	—	8 years after the disease recurred in the axilla, the scar remaining healthy. There was a large, hard, fixed axillary tumour, undoubtedly scirrhus cancer.	I saw this case at "consultations" with Mr. Bennett at St. George's on



43	A woman, aged 73, had the right breast removed for carcinoma at the "Cancer Hospital," Brompton, in 1885. The axilla was opened, judging by the scar	Case undoubted	In 11 years after, in 1896, she was in St. George's Hospital with a large recurrent mass of undoubted cancer in the axilla. This had been growing for about 2 years, so that she had remained free for about 9 years before the disease returned	Seen at "consultations" at St. George's, 1896.
44	In 1878 a lady, aged 68, was seen by Mr. Bryant with carcinoma in the cicatrix of a wound made for removal of a cancerous breast no less a time than 30 years before	The daughter, aged 50, had a hard cancer the size of an orange in her breast	The patient had remained well for 25 years before return took place. Then a tubercle appeared in the scar and other tubercles in the skin round it	Bryant's Diseases of the Breast, p. 158.
45	A woman, aged 50, was operated upon for cancer of 2 years' standing. The date is not given	Statement by Mr. Bryant	She remained well for 11 years, then local recurrence took place, which was removed. 2 years later she was well	Ibid., p. 221.
46	A patient, aged 67, had her breast removed for carcinoma in 1851 by Birkett	—	She remained well for no less a period than 23 years. At the age of 80 cancer recurred in the scar and opposite mamma	"
47	A patient, aged 30, had the breast removed by Birkett	—	She remained well for 13 years, then disease recurred in the scar and was removed. 2 years later another recurrence was removed	"
48	In 1866 Mr. Hilton removed the left breast for cancer from a woman aged 48. The axillary glands "were not looked for"	Statement of operator that disease was carcinoma	In 1876, 10 years after, this patient came under Mr. Bryant's care with cancer of the right breast, which was operated upon	"
49	A woman, aged 47, was one of a family of six sisters, five of whom and the mother had suffered from cancer of the left breast. In December, 1846, Arnott removed a tumour as big as the "last joint of the thumb" from near the nipple. "It was an example of genuine stony cancer"	This operation was doubtless incomplete, only the bulk of the mamma being removed	She remained well until 1856, when, 10 years after, there was a return in the scar and axillary glands. Mr. Shaw of the Middlesex Hospital removed them, and found to the naked eye and the microscope well-marked carcinoma	Morriss, Med.-Chir. Trans., 1867.

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
50	<p>In 1837 a lady, aged about 28, noticed a tumour of the right breast. She was pregnant at the time, and as she suckled her child it rapidly increased. In 1838 whole breast removed. In 5 months recurrent nodule removed from scar. In 1839 repeated removals by caustics by Brodie and Travers. Supposed to be cured, but again recurred. In 1842 very severe removal by caustics by M. Canquoin in Paris; subsequent healing. In 1843 sound healing occurred. Practically she had been under constant treatment for 6 years with caustic, and had sustained horrible sufferings</p>	<p>No doubt was expressed of the cancerous nature of this growth. There were many hard "tubers," and great fungation. It may possibly have been a recurrent sarcoma however, but all points to carcinoma</p>	<p>This lady died in 1892 of cardiac failure and pulmonary congestion at the age of 82. She had no trace of disease of the breast for 49 years. Singularly enough her daughter died of undoubted carcinoma of the mamma just after her mother succumbed to old age</p>	<p>Dr. Cullingworth (Med. Times, 1881, vol. i); also a medical relation of the family.</p>
51	<p>A lady, aged about 40, had the left mamma removed for undoubted carcinoma in 1889. Six months after the disease appeared in the opposite breast. This was also removed</p>	<p>There was no doubt that the disease was genuinely carcinoma, verified by microscopic examination as regards the first tumour. The axillary glands were not interfered with in either operation</p>	<p>In the year 1897 this patient is quite well without any return of disease, a period of 8½ years</p>	<p>Mr. Thomas P. Pick.</p>
52	<p>Breast removed from a lady, aged 49, in December, 1880. Three enlarged axillary glands also removed</p>	<p>Tumour the size of a large marble implicated the skin, which was freely removed</p>	<p>In 1886, 6 years after the operation, the cervical glands became implicated on both sides, the scar and axilla remaining sound. She died in 1888</p>	<p>Mr. Teale.</p>
53	<p>Breast removed from a lady, aged 45, in December, 1880. Three enlarged axillary glands also removed</p>	<p>Tumour was the size of a marble, and much skin was implicated</p>	<p>She remained perfectly well until March, 1889, then 9 years after the operation she died of glandular and visceral recurrence</p>	<p>"</p>

54	Removal of breast from a lady, aged 40, in December, 1880. Axilla opened, but no glands removed	Whole mamma and skin very freely removed	In 1887, 7 years after the operation, recurrence took place in the sternum, and was progressing towards a fatal end at last report	"
55	Removal of breast from a lady, whose age is not given, in June, 1884. Three axillary glands removed	Case perfectly obvious	In 1893, 9 years after the operation, she died of local recurrence	"
56	Removal of breast from a single lady, age not given, in October, 1884. Axilla opened, but no glands removed	Marked scirrhus implicating the skin	In 1893, 9 years after operation, a small nodule was excised from near the scar, about 1 inch away. The scar and axilla were sound. In 1896, 12 years after first operation, she succumbed to cancer of the lung	"
57	A woman, aged 60, had the breast removed by the late Mr. Hulke in the Middlesex Hospital in 1891. "No enlarged glands were felt in the axilla," which sounds as though that space was not cleared	Mr. Kellock, the registrar, details the characteristics of the tumour, which was undoubtedly carcinoma	In 1895, 4 years after the operation, this woman began to suffer from uterine hæmorrhage, and the uterus was successfully removed by Dr. A. Routh, who found carcinoma of the body of that organ. The breast scar remains healthy. She is now (1897) apparently well, 6 years after breast operation	Dr. Amand Routh; Mr. Kellock, Surgical Registrar of Middlesex Hospital.
58	A carcinomatous breast was by permission removed by the "cancer curer" Fell in the Middlesex Hospital. The specimen shows the whole mass destroyed and separated with a large bulk of skin	As the case was selected for a test of Fell's treatment we may be sure it was carcinomatous and a bad case	The patient remained well for 22 years, then recurrence of carcinoma occurred in the scar and proved fatal	Middlesex Hospital Museum series, xxx (2114).
59	A lady had a tumour excised from the right mamma in Feb., 1876, by a Portuguese surgeon in the Canary Islands. The growth rapidly recurred and was again removed. These two recurrences took place within 4 months. The third operation was performed by Mr. Willett, who removed the remains of the mamma and some enlarged axillary glands	Microscopical evidence of growth at third operation. Well-marked carcinoma of the chronic type	She died in 1885, having remained well for 9 years. Her death was certified as due to paraplegia and exhaustion. It is highly probable, therefore, that after 9 years the disease returned in the vertebral column	Mr. A. Willett.

No.	Primary operation.	Remarks.	Result as to recurrence and secondary operations.	Source or authority.
60	Left breast removed from a lady in 1885. Enlarged glands cleared from axilla	Microscopical evidence. Well-marked carcinoma of the chronic type	In 1893 a small recurrence took place under the pectoral muscle, a period of 8 years after the first operation. This was removed, and she had been well since	Dr. Whitwell, of Shrewsbury; Mr. A. Willett.
61	Amputation of left breast and clearing of enlarged glands from the axilla in June, 1891	Microscopical hard carcinoma	Well for 4 years, then recurrence took place in axillary part of scar. This was removed in March, 1895	Mr. J. Newland, of Clifton; Mr. A. Willett.
62	Right breast only removed for undoubted carcinoma in May, 1889	Clinical and microscopical proof	Absolutely well until 1895, a period of 6 years, when a gland began to enlarge in the axilla. This was removed with several others, and they were carcinomatous	Drs. Shipman and Wilson, of Grantham.
63	In 1880 carcinoma of the breast removed from a single woman, aged 45. Some cancerous glands removed from the axilla also	No doubt about case	In 1882 a secondary nodule was removed from the cicatrix. The patient remained well for 10 years, then in April, 1892, an enlarged gland was removed from the axilla, and also in September, 1892. In October, 1893, there was no sign of recurrence in the breast or side, but the patient rapidly succumbed with all the signs of intrathoracic cancer	Dr. Walker, of Peterborough.
64	In 1888 a hard nodular cancer of 7 years' duration was removed. The whole breast, some cancerous glands, and portions of the pectorals were removed, the operation being a very "complete" one. The patient was a short, stout woman, aged 57	Case quite undoubted. The cancer	She remained well for 6 years, then recurrence took place in the axilla. Now in 1897 it is extensive	Mr. John Murray, Middlesex Hospital; Mr. Roger Williams, of Preston.

(For Report of the Discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, pp. 35, 44, 56.)

# REMOVAL OF THE ENTIRE UPPER EXTREMITY FOR RECURRENT CARCINOMA

AFTER REMOVAL OF THE BREAST

BY

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IN 1890, Mr. T. F. Chavasse read a paper<sup>1</sup> before this Society on "Successful Removal of the Entire Upper Extremity for Osteochondroma." The operation, however, has so very rarely been performed for recurrent carcinoma after removal of the breast, and involves such different questions, that I venture to bring the following case under the notice of the Fellows.

S. T—, aged 53, single, a nurse, was admitted to St. George's Hospital in December, 1894. For about two years she had noticed in the left breast a lump which gradually increased and was painless. About June, 1894,

<sup>1</sup> 'Med.-Chir. Trans.,' vol. lxxiii, p. 81.

the skin over the tumour became adherent to it, and the growth commenced to increase rapidly. A week before admission ulceration commenced over the adherent patch. When admitted there was a well-marked scirrhus tumour occupying the centre and outer part of the breast. The nipple was not retracted; the growth was freely moveable over the deeper parts; the skin was ulcerated at the outer part, and infiltrated for some little distance around the ulcer. The glands in the axilla were found to be involved.

The breast and superjacent skin were freely removed. The fascia was dissected off the pectoralis major, and all diseased structures cleared out of the axilla. Under the microscope the growth presented the appearance of a carcinoma.<sup>1</sup> The patient made a rapid recovery, and left the hospital about a month after admission.

In September, 1896, *i. e.* two years subsequently, she was again admitted with a nodule about the size of a walnut situated just above the middle of the cicatrix. There was considerable pain in the growth and pain at times down the arm, with occasional swelling of the arm. The nodule was freely removed, together with a considerable portion of skin; and she again made a rapid recovery. Dr. Rolleston reported that the growth consisted of a carcinoma showing a transition from columnar-celled (or duct cancer) to a spheroidal-celled type.

Early in October, 1897, I received a communication from Dr. Maidlow, of Ilminster, informing me that the patient was suffering from recurrence of the growth high up in the axilla, associated with much pain and swelling of the arm. He suggested to me that the case might very probably be suitable for amputation. It is to his prompt recognition of the only kind of treatment likely to be of real benefit to the patient, before the

<sup>1</sup> In view of the subsequent history I regret that I cannot furnish a more detailed description of the specimen. Unfortunately, though preserved, it cannot now be positively identified. It was, almost certainly, a duct carcinoma.

disease had spread too far to render any operative interference justifiable, that the success of the case, so far as it has progressed, is largely to be attributed.

The patient, whose age was now fifty-six, seemed in fairly good condition. At the apex of the axilla, just over the main vessels, was an ulcerated surface with a hole in the middle large enough to admit the little finger. The skin about this point and the deeper structures seemed welded together. The skin around was a little infiltrated, rigid and shiny, as in commencing cancer *en cuirasse*, this condition extending some little way down along the thoracic wall. There was much pain in this situation and down the arm, with numbness of the fingers. The arm was greatly swollen, as can be judged from the illustration.

There was the usual distressing sensation of weight in the limb; still, though painful and swollen, she could use her hand to a limited extent. The scars of the previous operations were healthy. The skin over the pectoral muscle and clavicle as well as over the scapular and deltoid regions was sound. The clavicle appeared to be unaffected, but the disease extended right up to the scapula. There was no evidence of malignant growth in the mediastinum or elsewhere.<sup>1</sup> The soft parts, too, above the clavicle appeared to me to be free from disease; and, indeed, subsequently at the operation I could not discover that the supra-clavicular glands were affected. The case appeared to me to be one eminently suited to the removal of the entire upper extremity by what is commonly known as Paul Berger's operation.

On October 22nd I carried out this operation, following, save in certain details to be mentioned presently, the method advocated by Berger. The clavicle was some-

<sup>1</sup> In view of the subsequent history of the patient (see Table A, *infra*) I may note that there was no evidence of abdominal disease. Very probably there was really some malignant deposit in the liver at this time; but there was no symptom, physical or other, pointing to its existence. Hepatic cancer at times develops with astonishing rapidity.

what prominent, and I was able to cut down on this bone and saw it through, just external to the attachment of the sterno-mastoid muscle, very easily, an elevator having previously been pushed under the bone to protect the deeper parts. The clavicle was then forcibly lifted and cut through with bone nippers a little internal to the coracoid process. A greatly dilated vein at once came into view, almost of the size that one would expect the subclavian to be; it proved, however, to be the supra-scapular. This was tied in two places and divided, as were also the supra-scapular artery and the transverse cervical vessels. Some difficulty was experienced in securing the subclavian artery. This vessel was ligatured in two places with kangaroo tendon, and divided between the ligatures. I expected, as had happened to others, to have trouble with the subclavian vein, but the chief difficulty was to recognise the vein at all, for it was exceedingly small; evidently the greater part of the blood from the limb had to return through the supra-scapular. Scarcely any blood was lost during this part of the operation. Owing to the condition of the skin on the thoracic wall of the axilla, I had to make the anterior flap much smaller than is usually recommended, for it was judged advisable to remove a triangular portion of the skin directly below the axilla some four inches in length, and about the same width above at its base. The incision for the anterior flap, therefore, was not carried down to the inferior angle of the scapula, but stopped short on the thoracic wall vertically below the apex of the axilla. The flap was dissected up and the pectoralis major divided well internal to the pectoralis minor. This part of the operation was almost bloodless. The brachial plexus was cut high up, and the arm, on being rotated outwards, fell well away from the side. Next the posterior flap was made. This extended well over the deltoid region and posterior fold of the axilla, and joined the terminal incision of the anterior flap on the thoracic wall. The



scapular muscles were very easily divided; in fact, to some extent they seemed to tear away. The whole limb was drawn directly backwards during this part of the operation, so that the anterior surface of the scapula was entirely exposed to view just before the limb was actually detached. I had been prepared for hæmorrhage during this part of the operation, but the bleeding was most insignificant. Throughout the operation the greatest precautions had been taken to keep the patient warm, and especially to keep the wound warm. The moment the anterior flap was made, the exposed surfaces were covered over with hot sterilised towels, and the same precaution was observed with the posterior flap. There was little delay in securing the few vessels which it was thought prudent to tie. At no time during the operation did the patient present any symptom in the least degree alarming, and Dr. Dickson, who gave the anæsthetic, assured me that from first to last he had not a moment's anxiety. Probably not more than two or three ounces of blood were lost.

The patient was removed from the operating theatre in excellent condition; but, as so often happens after extensive operations, some five or six hours later the temperature dropped, the pulse became feeble, and for an hour or two she showed symptoms which are best described under the name of "shock." The condition, at no time at all alarming, rapidly passed off, and she slept well. The progress of the case was quite uneventful. A drainage-tube which had been inserted into the lower part of the wound was found lying loose in the dressings when these were removed on the fifth day. There had been a little oozing of blood. Nearly the whole of the wound healed by first intention, but from the upper part there was a slight discharge which commenced a week after the operation. The patient had disturbed her dressings here, and the wound might very easily have got septic. In all probability, however, the discharge

was due to a little sloughing of the end of one of the cut muscles.

On the sixteenth day she was able to get up, and she left for a convalescent hospital with the wound healed on the twenty-sixth day.

My colleague, Dr. Rolleston, has kindly furnished me with the following details of the dissection of the limb and the microscopical appearances.

"After removal, dissection showed that the ulcerated surface was continuous with a hard mass of growth which encircled and compressed the brachial vessels together with the median, ulnar, musculo-spiral, and musculo-cutaneous nerves. The brachial artery, however, was quite pervious. Microscopically the base of the ulcer was seen to be composed of carcinoma, in parts columnar-celled but in parts becoming spheroidal-celled; the growth showed some extravasation of blood into its substance. The coracoid process of the scapula was softened and infiltrated by white growth. Microscopic sections showed that the bone was completely invaded by a columnar-celled carcinoma, there being no transition, as in the ulcer, towards a spheroidal-celled growth.

"There were no growths in the clavicle or on the remaining part of the scapula, and there was no evidence that any growth abutted on the line of the amputation incision, so presumably all the growth had been removed.

"The arm was very œdematous, but there was no sign of any gangrene or necrosis.

"The chief points of interest from a pathological point of view are—

"(1) The free recurrence of duct carcinoma. In this case the growth recurred twice, and on the second occasion it infiltrated the coracoid process. Mr. Bowlby ('St. Bartholomew's Hospital Reports,' vol. xxiv, p. 272) concludes from an examination of seven cases, in none of which were the axillary lymphatic glands affected, 'that the axillary glands are not usually affected,' and that though the tumours are liable to recur locally they

are not so prone to affect the glands or disseminate as are the spheroidal-celled carcinomata. Secondary growth in bone has very seldom been recorded; Mr. Shattock ('Path. Soc. Trans.,' vol. xxxix, p. 324) has described a secondary growth of duct carcinoma in a rib.

"(2) The transition of type from columnar-celled to spheroidal-celled carcinoma, and the association of this structural character with free recurrence; in other words, as the histological characters passed into those of ordinary spheroidal carcinoma, the clinical features of duct carcinoma became replaced by those of ordinary spheroidal-celled carcinoma of the breast. This transition is commonly seen in duct cancer, and was well shown in Mr. Lunn's case of duct cancer in a man aged ninety-one ('Path. Soc. Trans.,' vol. xlviii, p. 247)."

So far as I have been able to ascertain, this is the fourth case in which this "interscapulo-thoracic amputation" has been carried out for recurrence of malignant growth after removal of the breast. Previous cases are one by Domenico Morisani and two by Mr. Treves.<sup>1</sup>

There are but few points that I need draw attention to as regards the details of the operation, for little can really be added to M. Berger's admirable description.<sup>2</sup> The most important point is to guard against any serious hæmorrhage; and this can best be effected by deliberation in the first stage of the operation. Not only is hæmorrhage prevented by tying and dividing the branches of the thyroid axis as soon as recognised, but by this step access to the subclavian artery is rendered much more easy. With regard to these smaller arteries I believe it to be better, for the sake of saving time, not to use the aneurism needle, but to clip them in two places, and then, after dividing the vessels between the two clips, to tie on the proximal side of the one and the distal of the other.

<sup>1</sup> See Tables, *infra*.

<sup>2</sup> Berger, Paul, 'L'amputation du Membre supérieur dans la contiguité du Tronc,' Paris, 1887.

In the same way, if the cords of the brachial plexus get in the way they are best divided at once, so as to expose more readily the subclavian artery. Moreover, after division of the pectoralis major, as the limb is rotated outwards the cords of the brachial plexus are put very forcibly on the stretch. Now stretching nerve-trunks of such a size unduly will, as is well evidenced in the case of the sciatic nerve, often produce a very marked lowering of the pulse, and I think on that account that the earlier the cords are divided the better for the patient. I made no attempt to divide the clavicle subperiosteally. This appeared to me to be unnecessary, as the vessels beneath were not endangered and the proceeding must occupy some time without having very distinct advantage.

In my case there were special reasons for absence of hæmorrhage. In the first place, at the previous operation many vessels had been tied in the axilla, though as these came off chiefly from the axillary artery the fact was not of much importance. Owing to the weight and pain of the limb, too, the patient had kept her arm at rest, and so lessened the supply of blood. The mass of cancer high up in the axilla had entirely encircled the axillary vein and materially diminished the calibre of the vessel, thus explaining the difficulty we experienced in recognising the subclavian vein, and also accounting for the dilatation of the supra-scapular. The operation, then, in cases of the kind I have described, is infinitely less formidable than for the conditions in which, in the great majority of recorded instances, it has been undertaken. Apart from primary amputations for injury, nearly all the cases have been for chondroma or for some form of sarcoma involving the bones of the shoulder-joint.

As regards the likelihood of hæmorrhage and shock and the prospect of recovery from the mere effects of the operation, patients with recurrent carcinoma in the axilla are in much better condition than those in whom the operation is undertaken for sarcoma. In the latter the subclavian vessels may be displaced and the vein may

very easily be injured. The vessels cut, too, are likely to be numerous and enlarged. If the growth is accidentally cut into or lacerated in manipulating the limb, the bleeding will be serious and difficult to control. The skin is stretched and thin over a large sarcoma, so that it is difficult to preserve the right amount. In cases of recurrent scirrhus, on the other hand, the flaps can be fashioned to a nicety; and, as good pressure can be easily applied, the ready union of the deep parts can be almost certainly secured—a point, of course, of far greater moment than the primary linear union of the cut edges of skin.

The superficial area of the wound, when the operation is performed for extensive sarcoma, must be very great, and there is serious liability to shock owing to chill. In my case the wound was as limited in extent as it is ever likely to be in this operation, and yet I estimated that the area of the moist cut surfaces from which evaporation and consequent chilling could take place, measured some 200 square inches. The absence of bleeding enabled us to keep the whole of this area warm throughout, a practical point of the first importance. Not only does the maintaining of the normal temperature of the tissues exposed in an extensive wound obviate the immediate shock, but it also prevents the serous exudation which, coming on a few hours after an operation, so exhausts the patient.

As regards the severity of the operation and the immediate risk to life, I consider removal of the entire upper extremity (implying by this removal of arm, scapula, and some part of clavicle) for recurrent scirrhus as less serious than disarticulation at the shoulder-joint for malignant growth of the humerus, and, *a fortiori*, far less formidable than the complete operation for sarcoma involving scapula and humerus.

Amputation through the shoulder-joint has been performed pretty frequently for recurrent scirrhus, though but few recorded cases can be found. The object has

been to relieve the patient of the heavy, swollen, and painful limb. In this object the proceeding may have a large measure of success, as the following case shows. The patient was under the care of Mr. T. R. Jessop, who has most kindly permitted me to mention the case. I give the account in Mr. Jessop's words:—"The woman, aged 56, came under observation in October, 1887, with extensive scirrhus in the left breast and a mass of axillary glands. I removed the whole. In August, 1888, I removed other glands from beneath the pectoral muscle. In January, 1890, I again removed diseased glands from the axillary and subpectoral regions, and in doing so the axillary vein was wounded and tied. In March, 1890, I amputated the arm (which was largely cedematous, livid, and brawny) at the shoulder-joint, obtaining good cover from the deltoid region, and at the same time I removed most of both pectoral muscles and subjacent tissue, all largely infiltrated with cancer. The scapula and clavicle were left. The amputation healed, and the patient remained in a satisfactory condition for about two years, after which she became bedridden and too ill to come here from her distant home, and finally died within three years of the amputation, presumably from return within the chest, seeing that she was reported to be 'very short of breath and unable to move in bed.'"

I am not concerned now either with advocating or criticising amputation through the shoulder-joint as a palliative measure in recurrent carcinoma. Cases in which this proceeding is indicated must be rare. My patient certainly did not fall into this class. Had I adopted this operation I should probably have cut into the growth while leaving untouched the source of pain, viz. the compression of the nerves. The removal of the heavy limb would have given no relief, for the pain would still have been felt in the arm and would have got worse. The complete removal rendered it possible to take away the whole of the obvious disease while cutting wide of its apparent limits. It was not merely a pallia-

tive measure. The reasonableness or, if the term be preferred, the justifiability of the operation was really identical with that of the original removal of the breast. Against recurrence no guarantee could be given then, nor can any be given now. But if operation was reasonable and proper in the original disease, it was also reasonable and proper in the condition of recurrence.

In defending the operation I am not anticipating but answering criticism. M. Paul Berger in his admirable monograph speaks very strongly, and considers that the operation should only be undertaken for suitable cases of malignant growth about the shoulder. Apart from traumatic cases, M. Berger enumerates but two in which the amputation was performed for other pathological conditions. In one the patient suffered from caries of the scapula, destruction of the shoulder-joint, and osteomyelitis of the humerus.<sup>1</sup> The man (aged twenty-three) recovered, and eight months later was reported entirely healed. In the second case the operation was undertaken for cancer of the breast.<sup>2</sup> M. Berger comments very severely on this case, not, it must be admitted, unjustly. The operation included removal of the breast and part of the second, third, and fourth ribs. The thoracic cavity was opened, and the patient died from shock in five hours. Possibly this case has done much to discredit the operation. At any rate, M. Berger does not contemplate the operation he describes for any such condition as that from which my patient suffered.<sup>3</sup> M. Berger seems

<sup>1</sup> M. Béranger-Féraud's case, 'Bull. de Thérap.,' Nos. 11 and 12, 1885. Quoted in Mr. Chavasse's tables, 'Med.-Chir. Trans.,' vol. lxxiii, p. 95. The clavicle was left in this case.

<sup>2</sup> Morisani's case, 'Il Morgagni,' Agosto, Ottobre, 1885. Quoted fully by Berger. Quoted in Chavasse's tables, 'Med.-Chir. Trans.,' vol. lxxiii, p. 96.

<sup>3</sup> "Nous ne trouvons plus guère d'autre affection pathologique qui puisse devenir pour l'amputation interscapulo-thoracique une cause raisonnable d'indication, . . . peut-être l'ablation d'un cancroïde fort étendu situé au moignon de l'épaule pourraient-elles conduire le chirurgien à cette extrémité. Nous ne pensons pas qu'il soit sage d'envisager sérieusement de pareilles hypothèses." Berger, *op. cit.*, p. 228.

scarcely to appreciate the excellence of his own work. By improving and rendering precise the details of the operation, he so reduced its immediate risk as to render it a recognised surgical procedure. Surely it is a little arbitrary to argue that the operation should be restricted to a single class of disease.

The objection to the operation on the ground of liability to recurrence in cases of carcinoma of the breast, loses much of its force if we contemplate the results of removal of the upper extremity for sarcoma. Mr. Chavasse tabulates forty-two cases of this nature.<sup>1</sup> Nine of these patients died from the immediate effects of the operation. Fourteen of the remaining thirty-three died from secondary deposits, eleven of them within a year of the operation. The liability to secondary growth in cases where carcinoma of the breast has recurred will almost certainly be larger, and the prospect of "cure" smaller. Against this consideration may be set the facts that the two classes of cases are not exactly parallel; that in recurrent scirrhus the relief from pain is likely to be the chief advantage secured; and that this relief is likely to be permanent. Mr. Treves unfortunately lost sight of his two patients after a few months, but both of them declared that the relief given by the operation made it well worth doing. My patient was of the same opinion.

Extensive operations such as the one under consideration have been denounced as "mutilations."<sup>2</sup> This form of criticism is rather like giving the method a bad name, and then condemning it. By "mutilation" is really meant, I suppose, disfigurement. The term conveys a reproach, and implies surgical wantonness. But the choice of the word is not happy when it is used to designate

<sup>1</sup> 'Med.-Chir. Trans.,' vol. lxxiii, p. 87, pp. 91 *et seq.*

<sup>2</sup> Thus M. Michon, in commenting on a case of removal of the scapula, communicated to the Académie de Médecine by M. Michaux, remarked, "J'aurais voulu que M. Michaux jugeât plus sévèrement ces mutilations que la chirurgie française proscribit absolument." Quoted by Berger, *op. cit.*, p. 189.



the removal of parts diseased, dangerous, painful, and in themselves disfiguring. The patient herself, unable to realise the relentless progress of carcinoma, will regard amputation of the breast for an apparently insignificant tumour as a greater "mutilation" than the removal of the whole upper extremity when the pain is acute, the limb swollen and useless, and the certain prospect of a slow and painful death looms large before her.

The disfigurement can be so completely concealed by an easily made appliance that it need hardly be taken into account. The shape of the shoulder can be restored more effectively than after amputation at the shoulder-joint, for in the latter cases the scapula and clavicle are drawn up, and the two sides of the body do not correspond.

It was perhaps unnecessary in my case to take away the whole of the scapula, for rough examination seemed to show that the growth had invaded only the coracoid process. There is little advantage in leaving part of the scapula, and it was judged safer to remove the whole bone. It seemed very probable that the disease had extended into the supra-spinous fossa. The arguments as to possible loss of blood did not apply, for there was no hæmorrhage from the posterior scapular vessels. Sawing through the neck of the bone protracts the operation, and would have been more than useless in my case, as the coracoid process was diseased. Moreover partial removal of the scapula is likely to lead to laceration of the muscles attached to the bone. Even if left the muscles are useless.

In the same way it might have sufficed to remove rather less of the clavicle. It seemed essential to remove some of the clavicle, considering the close relation of the outer part to the diseased scapula. Free access is wanted to the parts behind the clavicle. Further, the deformity will be less, and the flap more effectively adjusted if the clavicle is divided at the outer border of the sterno-mastoid muscle.

Although cases of recurrent carcinoma in which the

indications are favorable for this operation will but rarely be met with, I cannot but think that they will occasionally occur, and that the operation in such cases should be recognised as a very proper one to adopt.

Favorable indications would be—

(1) Previous removal of the breast with dissection of all the lymphatic tissue and fascia off the pectoralis major. The likelihood of mediastinal growth is greatly lessened if this method has been adopted, and if the cicatrix is healthy.

(2) Slow growth of the original tumour.

(3) Slow growth of the recurrent disease. The term "recurrence" is, of course, in many instances not scientifically accurate, but may imply only development and extension of disease that had not been detected at previous operations.

(4) Microscopic evidence that the carcinoma is of the columnar-celled or "duct" variety.

(5) Limitation of the recurrent growth, so that the incisions may be carried at least as wide of the disease as in removal of the breast and axillary glands for carcinoma of the mamma.

(6) The certainty of relieving pain. Unless the pain were severe, indeed the predominant symptom, the operation would hardly be taken into serious consideration.

The existence of a foul cancerous ulcer high up in the axilla might be taken as an additional argument in favour of the operation.

Contra-indications would be—

(1) The probability of leaving in the wound the cut surface of any part of the carcinoma. Rapid increase and fungation would be likely to take place.

(2) The involvement of the thoracic wall. Either the affected ribs would have to be left or the pleural cavity opened.

(3) The presence of an extensive degree of cancer *en cuirasse*. Although the existence of this condition does not signify that the whole of the hide-bound skin is

infiltrated with carcinoma, it furnishes strong evidence that the deeper parts, and very probably the pleura, are invaded. The skin flaps, too, would be likely to slough.

The more obvious contra-indications, such as the existence of cancerous deposits in other bones or in the spinal column, need hardly be enumerated.

As to the time for operation, the main indications that should guide the surgeon are the amount of pain and the degree of swelling of the limb. The surgeon would hesitate to recommend, and the patient would hardly give her consent to the removal of the limb so long as it was moderately useful. In the vast majority of cases, by the time that the limb has become useless and the pain acute, the propriety of removing the whole upper extremity will be negated on other grounds.

In some of these patients the division of the brachial plexus high up in the neck might be considered.

Briefly expressed, the operation is a proper one when humanity dictates it, and when the local, surgical, and general conditions do not forbid it.

The following tables represent the result of a tolerably diligent search through medical literature. In addition, I made inquiries of nineteen hospitals, or, in other words, of about 100 surgeons engaged in active hospital work. I have to express my best thanks to the surgical registrars of these hospitals, and especially to Mr. Treves, who, as will be seen by the tables, has furnished me with three unpublished cases. For invaluable assistance in compiling the tables I am indebted to Miss A. M. le Pelley, M.B.

TABLE A.—*Removal of the whole Upper Extremity for Scirrhus of Breast or Recurrent Scirrhus.*

No.	Operator.	Sex and age.	Disease.	Date.	Method.	Result.	Reference.	Remarks.
1	Morisani, Domenico	F., 54	Carcinoma of breast, thoracic wall, axillary lymphatics, &c.	March, 1885	Removal of outer two thirds of clavicle, scapula, arm, and surrounding soft parts, and several inches of second, third, and fourth ribs; thoracic cavity opened	Death from shock in 5 hours	Il Morgagni Agosto, Ottobre, 1885	Quoted at length by Berger. Case included in his and also in Chavasse's tables.
2	Treves	F., 57	Recurrent scirrhus; severe pain and swelling of limb. Breast removed Aug., 1889	July 3rd, 1891	Berger's	Recovery in 3 weeks; great relief	Lancet, 1891, p. 1158	Patient lost sight of after a few months; ultimate result unknown.
3	Treves	F.	Recurrent scirrhus; severe pain and swelling of arm.	—	Berger's	Rapid recovery; great relief	—	Case not previously published. Patient lost sight of after a few months; ultimate result unknown.
4	Dent	F., 56	Recurrent duct carcinoma; cancerous ulcer in axilla; severe pain and swelling of limb. Breast removed Dec., 1894, for carcinoma of two years' growth; axilla cleared out. Recurrent nodule in cicatrix removed Sept., 1896	22nd Oct., 1897	Berger's	Left for a convalescent hospital in 26 days; wound healed; great relief	—	Evidence of abdominal growth, probably carcinoma of liver; wound and its neighbourhood healthy. Died April 10th, 1898.

Although not strictly germane to my subject, I have thought it useful to tabulate the following cases, as a continuation of the list furnished by Mr. Chavasse ('*Med.-Chir. Trans.*,' vol. lxxiii, p. 91). M. Paul Berger (*op. cit.*) gives a list of twenty-two cases in which the upper extremity was removed for disease. He gives another list of "amputations consécutifs," and of amputations for injury. Twenty of the twenty-two cases are quoted also by Mr. Chavasse in his table of forty-four cases. In a paper read before the Clinical Society on March 25th, 1898, Mr. Barling mentioned nineteen cases in which the operation had been performed since 1890. The mortality is evidently now very small, though it may reasonably be doubted whether the percentage of recoveries is quite so great as the records of published cases appear to indicate.

TABLE B.—*Removal of the entire Upper Extremity for Sarcoma, &c.*

No.	Operator.	Sex and age.	Disease.	Date.	Method.	Result.	Reference.	Remarks.
1	Delorme	M.	Sarcoma	March, 1892	Subperiosteal section of middle half of clavicle; double ligation of artery and vein with division; removal of scapula, half of clavicle, and arm	Patientable to go about in 3 weeks	Bull. de la Soc. de Chir. de Paris, 1892, p. 448	—
2	Dubar	M., 27	Osteo-sarcoma	July 11th, 1894	Section of outer third of clavicle; ligation of vessels; ablation of scapula and arm	Got up on 10th day; recovery unimpeded	Bull. de l'Acad. de Méd., t. 31, 32, 1894	—
3	M'Leod	F., 15	Sarcoma	March 23rd, 1891	"Skewer" operation; a stout packing needle 8 inches long passed from below upwards through cavity of axilla; a second needle of same size was passed transversely behind the scapula; an india-rubber cord was wound figure-of-8 fashion round projecting ends; clavicle divided at outer third; scapula and arm removed	April 8th, wound granulating up well	Lancet, 1891, vol. ii, p. 117, and Indian Med. Gaz., 1894	M'Leod mentions two other cases which died of hemorrhage and shock, and attributes the success of his case to the use of the skewers.
4	Hall	—	Sarcoma	Reported, Jan., 1896	Posterior skin flap; removal of scapula; tying of vessels; disarticulation of clavicle; removal of limb and tumour	Recovery in 5 weeks	Indian Med. Rec., 1896	—
5	Keen	F., 20	Myeloid sarcoma	Nov. 20th, 1893	Resection of middle third of clavicle; ligation of vessels; removal of scapula and arm	Sent home healed in 3 weeks	Trans. Amer. Surg. Ass., 1894	—



No.	Operator.	Sex and age.	Disease.	Date.	Method.	Result.	Reference.	Remarks.
14	Treves	F., 19	Myeloid sarcoma of upper end of humerus; shoulder-joint invaded. Disease began with "weakness in the shoulder in Nov., 1892." Swelling of upper end of humerus noticed Dec., 1893	June 6th, 1894	Berger's	Rapid recovery. Perfectly well and free from recurrence July, 1897	—	Humerus became fractured during operation. Case not previously published. Communicated to me by Mr. Treves.
15	Pearce Gould	M., 36	Round- and spindle-celled sarcoma of humerus	July, 1897.	Berger's	Left for convalescent home on 16th day; wound soundly healed	—	Case not previously published.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 101.)



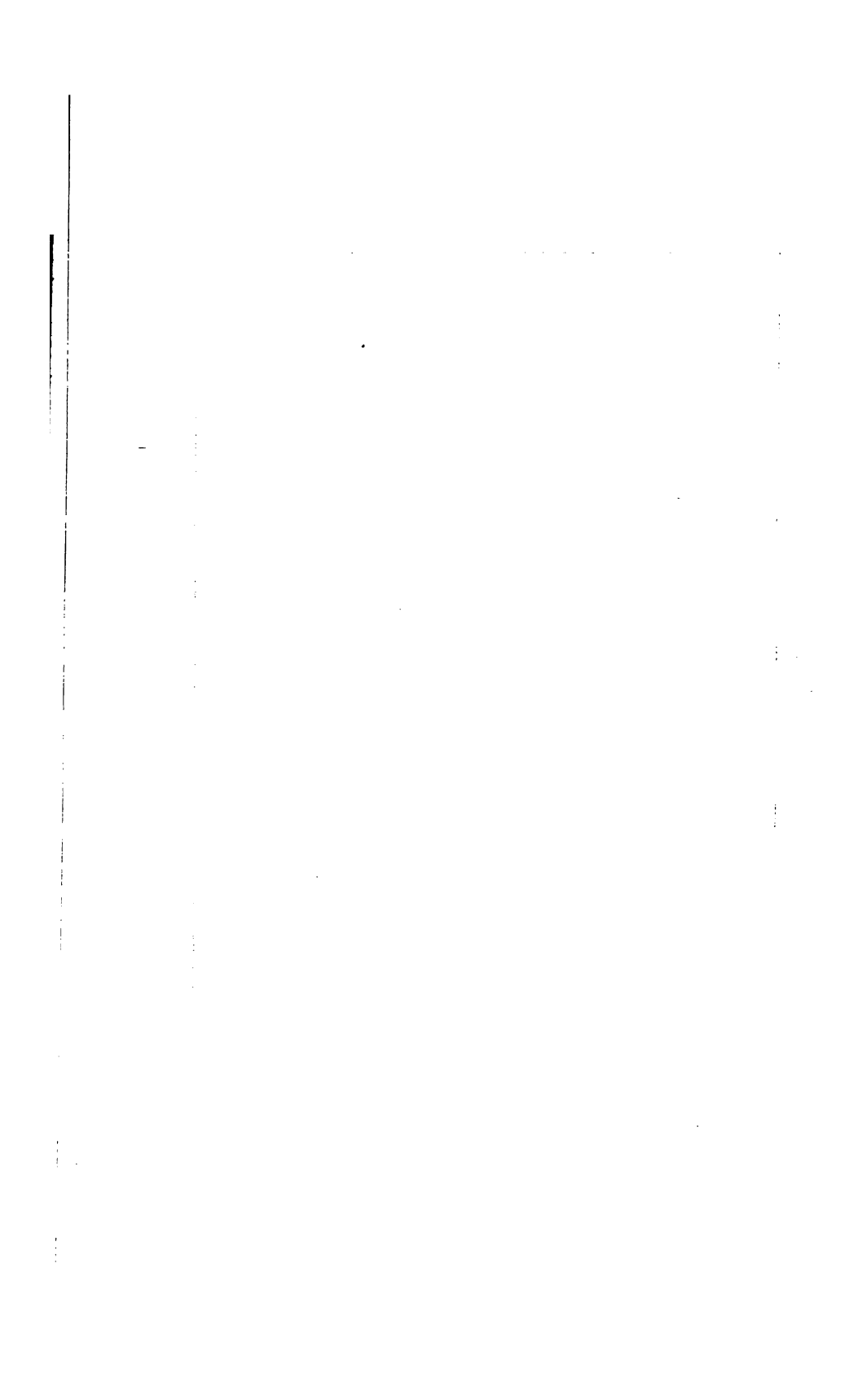


**DESCRIPTION OF PLATE IV.**

**Removal of the Entire Upper Extremity for Recurrent Carcinoma  
after Removal of the Breast (CLINTON T. DENT, F.R.C.S.).**

Recurrent carcinoma mammæ causing swelling of the arm, for  
which Berger's operation was performed.







DESCRIPTION OF PLATE V.

Removal of the Entire Upper Extremity for Recurrent Carcinoma  
after Removal of the Breast (CLINTON T. DENT, F.R.C.S.).

Result of removal of entire upper extremity (Berger's operation)  
for recurrent carcinoma mammae, three months after operation.







ON THE  
EPIDEMIC MALARIAL FEVER OF ASSAM  
OR  
KALA-AZAR

BY  
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THE disease known in Assam under the name of Kála-azár, or Black Fever, has excited attention in India during the last fifteen years. It was first described in the sanitary report of Assam for the year 1883, when it was mentioned as "a malarial cachexia produced by malarial fever, deriving its peculiar characteristics from the nature of the region where it prevails, and having a singular tendency to run rapidly into the cachexial stage." It was also recorded at this time that according to popular opinion the disease was contagious, and that large tracts of country were being depopulated by it, chiefly in the Terai region at the foot of the Garo Hills. During the next two or three years the disease

spread into the neighbouring district of Goalpara, and then into the Kamrup district still further to the east. Up to this time every medical officer who had seen the disease was of the opinion that it was malarious in its nature, but its steady spread eastward year by year, and its appalling mortality, made some doubt whether there was not some other factor besides malaria in the disease. As anæmia caused by the *Anchylostomum duodenale* had also been recently found to be very prevalent on tea-gardens, more especially in the easterly parts of the Assam valley under the name of beri-beri, Surgeon-Major G. M. Giles, of the Indian Medical Service, was deputed by the Government to investigate and report on both diseases. He began work at about the end of November, 1889, and finding the anchylostomum both in cases of kala-azar and in the so-called beri-beri, while fever was in his experience an inconstant feature of the former disease, he came to the very natural conclusion that both diseases were essentially due to this worm, and only differed in that kala-azar was more frequently complicated by malaria. During the rainy season he worked in Shillong, the hill station of Assam, at the life history of the parasite.

Unfortunately he omitted to examine healthy people for the parasite, and he only worked in the plains during the cold weather season, when, as I shall show presently, such cases of kala-azar as survive the rainy season lose their fever, only too often for it to recur again during the next rainy season, so that he never saw the disease during the season of its greatest activity. Moreover, it was subsequently proved, chiefly by Dr. Dobson, also of the Indian Medical Service, that over 80 per cent. of healthy coolies, imported into Assam from various parts of India, harbour the anchylostoma in numbers varying from units to hundreds. As the disease continued to spread slowly some ten or fifteen miles a year into new districts in a wave of increased fever mortality, it was decided to have the question re-investigated, and I was fortunate enough to be selected for the task, and com-

menced work in the Nowgong district in April, 1896, and presented my report just one year later.

The problem which presented itself for solution was as follows—(1) was the disease anchylostomiasis alone? or (2) was it purely malarial, and if so how was its steady spread to be accounted for? or (3) was it a combination of the two former, the presence of the intestinal worm accounting for the spread of the disease? or (4) was it wholly or in part due to some hitherto undiscovered cause? The third opinion, that it was a mixture of malarial fever and anchylostomiasis, was apparently the view which was most commonly held, at least outside Assam itself.

The first thing to be done was to try to find out what part, if any, the anchylostomum and malaria respectively played in the causation of the disease. It might appear at first sight that the fact which has been already mentioned, namely, that the anchylostoma are found in larger or smaller numbers in a large percentage of healthy natives of Assam and other parts of India, alone throws great doubt on their being the cause of kala-azar in the absence of evidence as to the numbers found in the latter disease; but it was argued by the upholders of the worm theory that even if few or no worms are found in the later stages of, or after death from, kala-azar, yet they may have been present in larger numbers in the earlier stages of the disease; and although it is generally allowed that about 500 of these blood-sucking worms must be present in the small intestine for from six months to a year in order to cause marked anæmia, yet it is very difficult to prove that large numbers of the worms have never been present in given cases of kala-azar, which disease usually lasts from four to nine months, and may occasionally run on for two years. Fortunately a way of overcoming this difficulty was found in an examination of the type of the anæmia in the two diseases. It occurred to me when reading up the literature of the subject that an anæmia

produced by a steady drain of small quantities of blood day by day, such as is produced by the anchylostomum, would differ from that found in chronic malarial fever, in which the red corpuscles are destroyed in the blood by the amoeba of Laveran. As I was not able to find any very full account of the quantitative changes in the blood in these two diseases in the scanty literature within my reach, I determined to make an examination for myself with the aid of the hæmocyto-meter, hæmoglobinometer, and Lloyd Jones' instrument for estimating the specific gravity of the blood, with the result of showing that the anæmia of kala-azar differed so widely from that of anchylostomiasis that, given a drop of blood from typical cases of each disease, a correct diagnosis could be made. The following table shows the differences at a glance, and also the close agreement of the type of anæmia in the cases of kala-azar and that of ordinary malarial cachexia in a district which was not affected by this scourge, all being compared with the blood of healthy natives of Assam, as their standard differs greatly from the European one according to Sir Wm. Gowers' instruments.

The Blood in	Percentage of hæmoglobin.	Red corpuscles per cubic millimetre.	White corpuscles per cubic millimetre.	Ratio of white to red corpuscles.	Specific gravity of blood.	Hæmoglobin in each red corpuscle.
(1) Healthy natives of Assam .....	62	4,734,000	7,325	1:684	1·054	·65
(2) Kala-azar cases... ..	33·45	2,462,000	2,600	1:1,170	1·048	·65
(3) Ordinary malarial cachexia .....	31·6	2,000,000	1,600	1:1,400	1·042	·73
(4) Anchylostomiasis .....	15·2	2,145,000	5,338	1:524	1·034	·31
(5) Mixed cases of malaria and anchylostomiasis	27·4	3,120,000	3,200	1:975	1·039	·43

In the first line the average blood of healthy Assamese is given, from which it will be seen that although the

red and white corpuscles average about the normal number, yet the percentage of hæmoglobin averages only 62 per cent. of Gowers' standard, and consequently the average amount of hæmoglobin in each red corpuscle or the hæmoglobin value or colour index is only '65. I also ascertained the interesting fact that seven Europeans living in Assam only average 71 per cent. of hæmoglobin during the hot and rainy seasons, but improved by some 10 per cent. during the healthy cold weather.

The second line shows the average blood of a large number of cases of kala-azar, and it will be noticed that there is marked reduction of both the red corpuscles and of the hæmoglobin, but in equal proportions, so that the hæmoglobin value is the same as in healthy Assamese. The specific gravity of the blood averaged 1·048, being somewhat below normal; while the number of the white corpuscles are markedly reduced both absolutely and relatively to the red, so that the average proportion was 1 white to 1170 red.

The third line shows the average condition of the blood in cases of ordinary malarial cachexia in a part of Assam which was unaffected by the epidemic disease, and it will be observed that in all respects it resembles that of the kala-azar cases. The figures given by Cabot in America, and Waddell in India, confirm my results on this head.

The fourth line shows the average blood-state of cases of anchylostomiasis examined by me. It will be seen that the anæmia in these cases was more extreme than in those of kala-azar, but of much greater importance is the marked difference in its type. The differences are, firstly, in the relatively greater decrease of the hæmoglobin as compared with the reduction of the number of the red corpuscles, so that the hæmoglobin value only averages '31 or less than half what it was in kala-azar; secondly, while the white corpuscles are absolutely reduced in numbers, relatively to the red they are increased, being in the proportion of 1 to 524; and thirdly, the

specific gravity of the blood is reduced to the very low average of 1.034, and it may at once be said that these differences are so uniform in different cases that the two diseases can be completely differentiated by their means. Sandwith, of Cairo, also found a very low hæmoglobin value in 173 cases of anchylostomiasis. In the last line is shown the average blood-state of four cases in which kala-azar was complicated by considerable numbers of anchylostoma (from 80 to 120), and it will be seen that the figures are intermediate between those of the two primary diseases.

The differences found are, moreover, just what might have been expected, for in the case of chronic malarial fever the hæmoglobin is not lost to the body, but is stored up in the liver, spleen, &c., in the form of a pigment containing iron, which can be utilised to re-form hæmoglobin to stock the red corpuscles which are being formed in the bone marrow, so that the hæmoglobin value of each corpuscle does not fall. It may be mentioned here that I found the yellow marrow in the shafts of the long bones to be constantly converted into red marrow in fatal cases of kala-azar, just as occurs in pernicious anæmia. The white corpuscles become gradually reduced in the course of their long fight with the malarial parasites, but a temporary relative leucocytosis occurs during actual fever. On the other hand, in anchylostomiasis the constant drain of hæmoglobin from the body, and the great difficulty in replacing it, causes the hæmoglobin value to fall as soon as the reserve iron in the liver becomes much reduced, as is very soon the case. The losses of the red corpuscles, and still more those of the white, are more easily replaced, and their averages remain comparatively high. The constant loss of albuminous and saline substances as well as of the hæmoglobin accounts for the remarkable diminution of the specific gravity of the blood in anchylostomiasis.

The above-described difference of the type of the anæmia found in kala-azar from that which is caused by

anchylostomiasis, together with the facts that in 83 per cent. of kala-azar cases I found less than twenty of the worms present during life—a number which I proved to have no effect on the blood of healthy natives,—and that there were fewer anchylostoma found post mortem in twenty-five fatal cases of kala-azar than were present in those who had died from accidental causes; prove conclusively that anchylostomiasis is not the cause, nor even a contributory cause, of the Assam scourge which is under discussion.

Having thus cleared the ground, it became necessary to determine the part played in the causation of the disease by malaria. I have already shown that the blood-changes are those of ordinary chronic malaria. Moreover I found that during the whole of the hot weather and rainy season, lasting from March to October, fever of an intermittent or remittent nature is present in this disease, with occasional intermissions of from a few days to a few weeks (see temperature chart). Again, the anæmia progressively gets worse during the fever periods, and improves somewhat in the intervals of apyrexia. The cases usually last from four to nine months, and are rarely fatal in less than three months; but with long intervals without fever in the dry cold weather the disease may last two years. Briefly, the disease is characterised by more or less constant intermittent or remittent fever, with marked general wasting of the body, but with great enlargement of the spleen and liver—the former organ averaging over 2 lbs. in weight post mortem,—accompanied in a minority of the cases by œdema of the feet, and always with more or less severe anæmia, great weakness, and often a darkening of the skin, from which the disease probably gets its name of black fever. Death occurs either from exhaustion due to the fever, or from a chronic form of diarrhœa due to atrophy of the mucous membrane of the small intestines and consequent loss of digestive power, or if the patient survives until the onset of the cold weather, pneumonia and other lung

diseases often carry him off. The general condition of the patients is well seen in the photographs which I pass round, and some of which are in my report. The seasonal incidence of the disease is of great importance, as I find that the great majority of patients begin their illness during the rainy season, especially from April to August, and during the dry cold months from November to February very few new cases occur. The season is that of the ordinary malarial fevers of Assam, only it is a somewhat more extended one.

The disease, moreover, is quite indistinguishable in its early stages from the ordinary malarial fever of Assam, and in its later stages it exactly resembles ordinary malarial cachexia. It is, however, characterised by its very great intensity, as shown by the fact that it may produce marked cachexia in as many months as the ordinary malarial fever of unaffected parts of Assam often takes years to produce, while it is very resistant to small doses of quinine, and is of so persistent and virulent a nature that among 200 cases on a tea-garden, where the cases had been treated throughout by an experienced European doctor—Mr. J. Dodds Price, to whom I am greatly indebted for much valuable assistance during the course of my investigation,—the mortality was 96 per cent. The other marked characteristic was the way in which it attacked large numbers of a family, which I can best indicate by the fact that in a series of cases in which I made a note of every member of the patient's near relatives who was alive, and every one who had died of the disease, I found that three quarters of my cases had lost half or more than half of their whole household. I have also seen the last of a family of thirteen, the other twelve having all died of the disease.

But I must pass on to the search for micro-organisms in the disease. Dr. Giles had previously failed to find any organisms which could be cultivated from the blood, or which could be stained in any of the organs after death. I also inoculated blood from the finger-tip and that



drawn from the spleen itself during life on agar-agar with negative results. Moreover, sections of the various organs were stained for bacteria, by various methods, by both Dr. Giles and myself, with negative results.

On the other hand, I examined a large number of cases for the malarial organisms and had no difficulty in finding the amoeba of Laveran in all stages of the disease. As I expected to find some differences in those present in this peculiar form of fever from those of ordinary malaria, and hoped to be able to differentiate the two diseases by this means, a careful study of the forms met with was made in early stages of the disease, before marked changes had taken place in the blood. At the same time full notes were taken in shorthand of each case, and temperatures taken four times a day. A series of early cases of fever were thus examined, which could not as yet be said to be typical of kala-azar, but some of whom lived in houses inhabited by typical cases of the epidemic disease. After six or eight weeks they were all re-examined, and by this time many of them had recovered and were apparently only ordinary malarial fever, but others had become typical cases of kala-azar, with constant fever and cachexia. The notes and organisms of the two classes of cases were compared, but no definite differences could be detected. Moreover the organisms found in both groups were the ordinary forms, so well depicted in the plates in the Sydenham Society's translation of Italian works on malaria. They were also similar to those I had previously studied for many months in Bengal.

Again, the post-mortem changes were in all respects those of malarial cachexia, while there is not a symptom or a pathological change that I have found in kala-azar that is not met with elsewhere in malaria,—as, for instance, in the very full description of the disease as seen in Algeria by Kelsch and Kiener. Marked pigmentation of the liver, spleen, kidneys, &c., which is such a characteristic feature of malaria, was constantly found, which

under the microscope showed the same distribution as is figured in the work of the last-named authors, while an analysis of the amount of iron in the liver showed it to be increased. The disease, then, was entirely malarial in its nature, and only differed from ordinary malarial fever in its much greater intensity, its peculiar distribution in families, and its power of spreading.

The next point to be studied was the way in which the disease spreads from village to village and district to district, in order to determine if the disease is a communicable one; and if that proved to be the case, the origin of such a power in a malarial fever remained to be elucidated.

It will be well to say a few words on the ravages of the disease. It has already been mentioned that certain tracts of country have been depopulated by this scourge, but an idea of its ravages can be best conveyed by the figures of the census returns, and by the decrease in the amount of land under cultivation owing to the death of so many workers. Thus between the years 1881 and 1891 there was an increase of 19·36 per cent. in the inhabitants of that portion of the Goalpara district which lies north of the Brahmaputra River, and was very little affected by the epidemic fever; while in the affected southern part there was a decrease of 18·08 per cent. in the same period. In the Kamrup district, which was attacked only during the latter part of this decade, it was calculated that 75,000 persons died of this disease, out of a population of 644,960,—that is, nearly one eighth of the population; and here again the whole of the district was not attacked, and in the southern part there was a decrease of 12 per cent. in the population. In the Nowgong district, which has been attacked during the last decade, just about one fifth of the land which was previously cultivated is now untilled, with a corresponding loss of revenue, and the deserted tumble-down villages and patches of high jungle grass growing in recently cultivated fields testify more eloquently than any words to the terrible nature

of the scourge, which has probably carried off about one fifth of the population of an area extending over some two hundred and fifty miles in length, and varying from twenty to sixty in breadth, in the course of the last fifteen to twenty years. Moreover it is still spreading, with but little abatement of its virulence. It must, however, be mentioned that the disease passes over a district in the course of eight or ten years in a wave of greatly increased death-rate from fever, and then the mortality declines again, and the population of the Garo Hills, which was first attacked, is now once more on the increase.

The general course of the spread of the disease presents some points of interest. Thus it never spreads far up into the hills. The Garo Hills consist for the most part of low hills of from 500 to 1000 feet, with flat alluvial and marshy rice ground between them,—in fact, a typical terai. The higher central range reaches 4000 feet, but the disease affected this part but little. Again, the Kashia Hills, which run up to 6000 feet, were not invaded. The disease was for many years chiefly confined to a narrow tract of country between the hills just mentioned and the Brahmaputra River, and spread slowly eastward along the Grand Trunk road. In places where there was much traffic across the river it spread to the north bank, which with its many sandbanks is often five to ten miles in breadth; but as the more thickly populated parts on the north bank are separated from each other by large rivers and stretches of twenty miles or more of uninhabited jungle, it showed very little tendency to spread eastwards along the north bank of the river. When the disease reached Gauhati, a large town on the south bank of the river in the easterly part of the Kamrup district, with a great deal of traffic and intercommunication with other places east of it, the disease spread in different directions, but always along lines of communication. It now reached the wide district of Nowgong, and here it spread chiefly along the densely populated banks of the Kulung River *right away*

*from in under the hills ; yet, although it ceased to be in any way a terai disease, it was none the less virulent. In short the disease has always spread along the lines of human intercourse, and has ceased to spread when a tract of uninhabited jungle obstructed it. Moreover the parts affected all have an alluvial soil, and it fails to get a footing far away from this up in the hills.*

Coming now to the way in which it is carried from village to village, I may say as a result of walking several hundred miles, making inquiries in the affected districts, that the disease is carried by one person who has contracted the fever in an infected village, very often while on a visit to a relative, returning to his own previously uninfected village with the fever upon him, with the result that others of his household next are attacked during the ensuing month or two, after which the fever spreads to others in the village during the rainy season. During the next cold weather, when the ground is dried up, and little or no rain falls for three or four months, the cases of fever greatly decrease or disappear, only to break out again in the same villages and houses at the beginning of the next rainy season in March or April, when the ground is once more moist and warm, and so it goes on for from five to seven years until apparently all those who are susceptible have suffered from the disease, and not rarely as many as two thirds of the village have died, including a large proportion of the children. Nothing struck me so much as the way in which the ignorant villagers recognised the first cases of the disease that occurred amongst them as something much more virulent and fatal than the ordinary fever to which they were accustomed, and it became monotonous to hear the head man of a village, who registered the deaths, when asked how the disease began in his village, say, " Oh, such and such a man [naming him] came from such and such a village [which was an infected one] with the fever ; " he was the first, and his wife or one of his children was usually the second to suffer from it. Orphans whose parents had

died of the fever often introduced the infection into houses of relatives who took them in, either in the same or in a different village. Years ago the Garos were known to take a sufferer from the disease out into the jungle, make him comatose with drink, and burn him to death in the hut which they had erected for him, or else place food near him day by day and run away for fear of infection. I have myself seen a victim of the disease turned out from a village by her husband, while her own father was prevented from taking her into his house for fear of infecting the village in which he lived. Once more, I have seen a small village of Brahmins, who could hold no intercourse for caste reasons with those around them, remain free from the disease, although all the villages around them had suffered severely for several years. Again, in other cases I have seen one part of a village badly affected, while another part of the same village two or three hundred yards away, the people of whom had cut off all communication with the affected part, had entirely escaped. Such instances will be found recorded in my book, and they afford ample evidence that the disease is slowly communicable from man to man, either directly through the air, or more frequently, I think, after passage through the soil. But I must pass on to the origin of the epidemic. So far I had come to the conclusion that the disease was purely malarial, while it was certainly very intense and slowly communicable. It remained to be discovered how a malarial fever could have attained to such powers.

While looking up the literature of kala-azar before visiting Assam I was struck by its resemblance in its general features to the famous "Burdwan fever" epidemic of the fifties, sixties, and early seventies. In this case a very fatal form of an admittedly malarial fever spread from one district in Lower Bengal to another over a large tract of country for some twenty years, and carried off half a million of people in the Burdwan subdivision alone. I shall not discuss here the resemblances

of these two epidemics, as I have done so elsewhere ; but its observation made me at one time think that the two phenomena might be connected, and led to a study of the old Bengal reports which revealed the true origin of the mysterious kala-azar. In the first place I found that the so-called Burdwan fever was quite independent of the Assam epidemic, as it died out when it reached rising rocky ground on the west and north-west, and was checked when it encountered dry, porous, laterite soil in the south, and was never able to get any permanent footing away from alluvial soil.

On the other hand, I found there had been an epidemic of malarial fever in the early seventies in the Rungpore district of Bengal, which is only separated from the foot of the Garo Hills by the southward bend of the Brahmaputra River. This district is so very low-lying that it is to a large extent under water during the rainy season from June to September, and at this time the fever is at a minimum. When the country dries up after the rains are over, malarial fevers become very prevalent, and last for about three months. When the rains are steady and last late the autumnal fever has a short duration and low mortality ; but if the rains are short and unevenly distributed, so that the country, which is under water in July, partially dries up, say in August, the fever becomes very prevalent, and its season is much extended, with a resulting heavy mortality. Now between the years 1872 and 1877 there was an extraordinary succession of five out of six years of very deficient and unevenly distributed rainfall, such as has not occurred before or since as far as I have been able to obtain the records ; and this was accompanied by a fever mortality which became increasingly great each successive year, except the one in which the rainfall was normal, in which the fever rate slightly declined. The mortality was so great that there was a decrease in the population of the Rungpore district between the two censuses of 1872 and 1881, just as there was in each district of Assam which

was subsequently invaded. The fever was also recorded as having been more frequently remittent in character, and had a much longer seasonal prevalence, showing an increased intensity of the disease; and in 1875 it was noticed as being especially severe in six areas, all of which were situated on the eastern side of the district, including the low banks and islands of the Brahmaputra River, which takes it up to the foot of the Garo Hills.

Now, before I knew any of the details of the Rungpore outbreak, I visited the Garo Hills, and found from an examination of both the medical and administrative reports that the so-called kala-azar only appeared in an epidemic form, depopulating whole villages, in the year 1875, as evidenced by the fact that this was the first year in which the revenue, which took the form of a house tax, fell below the estimate, owing to the decimation of a group of villages by this disease, but from that date onwards its ravages have been recorded in the revenue reports year by year. Now this group of villages, and also another group fifty miles further north, which was attacked about the same time, are both exactly in the two lines of traffic between Rungpore and the foot of the Garo Hills, and the whole of the Assam epidemic has been traced year by year from this district, as is seen in the map which I show you, the figures in which represent the dates when each place was invaded by the epidemic. It will be noticed that 1875 is the very year in which the Rungpore epidemic fever was so bad on the banks of the Brahmaputra River. The inference obviously is that the epidemic began in Rungpore owing to an intensification of the ordinary malarial fever of that very malarious district, by the extraordinary succession of unhealthy years due to deficiency of the rainfall, until it attained to the power of spreading, and it then crossed the river and invaded the foot of the Garo Hills. Possibly the malarial organisms became intensified by the prolonged fever season allowing of their being rapidly passed through a

series of susceptible persons, but this point cannot be tested until the amoeba can be cultivated. The facts, however, remain, and the analogous cases of such diseases as pneumonia and pestis minor, becoming occasionally intensified into epidemic pneumonia and virulent bubonic plague respectively, may be adduced in favour of the view that malaria may become intensified by extraordinary circumstances (as it were in nature's laboratory) until it attains to the power of spreading to other districts in the absence of physical or seasonal changes in those districts to account for its breaking out in them, and only limited by the necessity of a suitable soil, such as an alluvial one. It is very difficult to prove the introduction and spread of an epidemic malarial fever in a district which is already notoriously malarious, but in the case of the outbreak in Mauritius in 1865, in an island hitherto free from malarial fever, the disease appears to have been introduced by coolies from India, and it produced such a fearful epidemic that one third of the population are said to have died in four years, and the disease remains in a milder form to this day. Now it is worthy of note that this occurred during the height of the Burdwan fever epidemic in Bengal, and in the very districts from which coolies are exported from India. Further, it was noted that the form of fever in each instance was very similar. Now there is a good deal of evidence to show that the Burdwan fever was slowly communicable, just as I have shown the Assam malarial fever epidemic to be, so that it appears to be possible that the Mauritius fever was an instance of one of these Indian malarial epidemic fevers being imported into an island previously free from malaria, and spreading with a fearful virulence. Now let it be observed I do not say that all malarial fever is communicable, but only that it may become intensified until it becomes so, as in the case of pneumonia, and as most pathogenic germs which can be cultivated may be artificially intensified. Other epidemics of malarial fever in various countries might be mentioned which were pro-



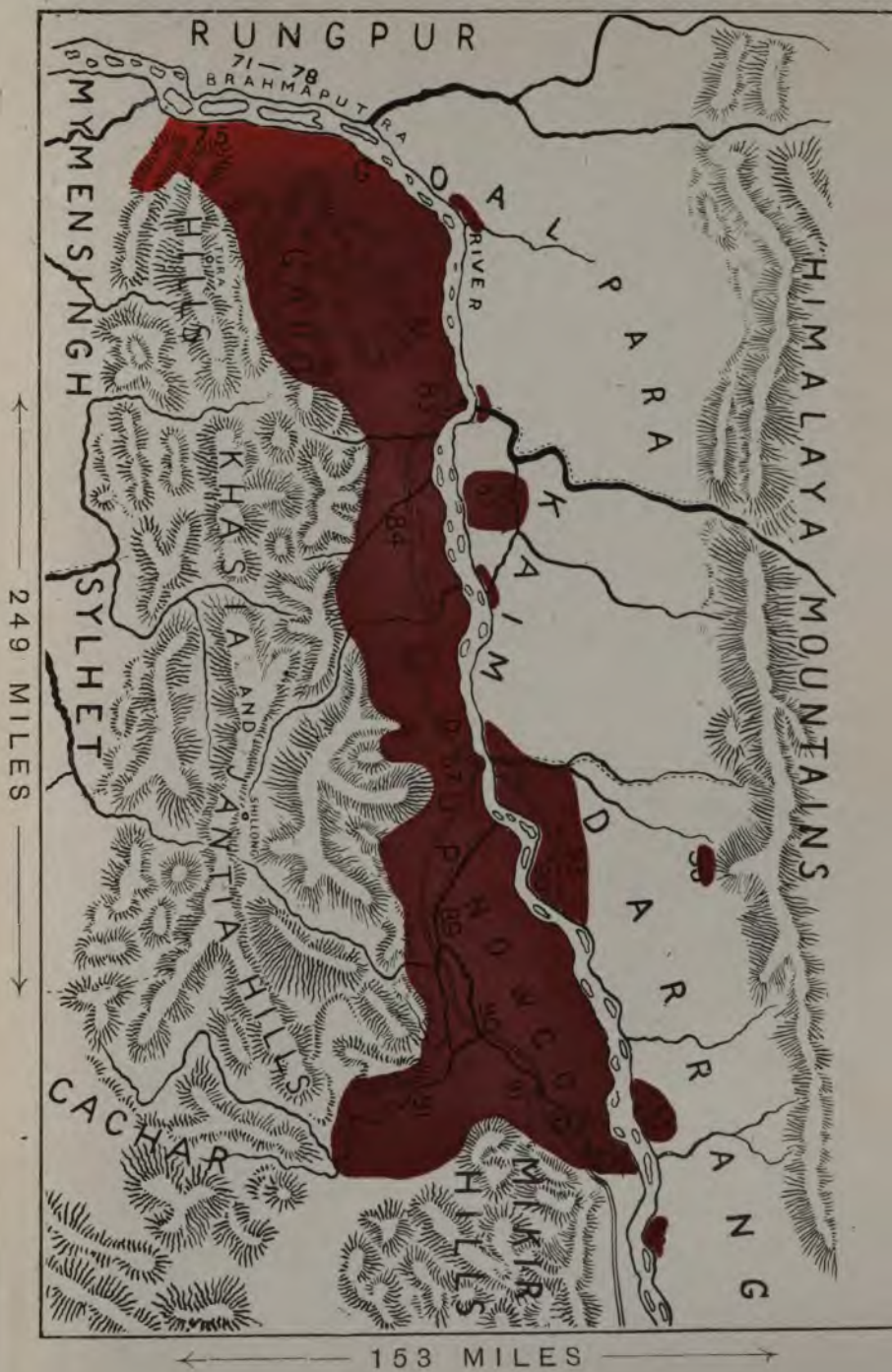
bably of a similar nature to the Assam epidemic, but space does not permit. As I have been fortunate enough to have had a unique opportunity of investigating such an epidemic malarial fever in the light of recent pathological and bacteriological knowledge, I thought a brief account of my conclusions might not be without interest to this Society.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 110.)

**DESCRIPTION OF PLATE VI.**

**On the Epidemic Malarial Fever of Assam, or Kala-azar  
(LEONARD ROGERS, M.D.).**

**The numbers refer to the years of prevalence, and the parts  
marked red to the distribution of the disease.**





A CASE OF ALBUMOSURIA

IN WHICH THE

ALBUMOSE WAS SPONTANEOUSLY PRECIPITATED

BY

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(COMMUNICATED BY DR. LAUDER BRUNTON)

Received December 20th, 1897—Read April 26th, 1898

THE case which I wish to bring to your notice is that of H—, a man 70 years of age, a small shopkeeper in Liverpool. He has enjoyed fairly good health for the greater part of his life, having never had any serious illness. He states that some years ago his urine was occasionally turbid, and that he had it examined by a medical man; but as he believes it became clear on heating, and the practitioner assured him that it was healthy, I conclude the turbidity was of a different nature from that observed now. His present complaint seems to

date from about the end of last year (1896), when he experienced a severe domestic affliction. He states that about Christmas he noticed something wrong with the urine—that it looked like coffee on one occasion, but the exact condition I am unable to ascertain. After this he noticed from time to time that it was turbid when voided, resembling the specimen I now show. This turbidity has been noticed at intervals ever since, but has gradually increased in frequency, so that at the time of writing (December, 1897) it is often present on several days in succession.

*Present condition.*—He is a spare, rather pale man, below the average height. He easily gets tired, and often suffers from pain and a feeling of weakness in the lumbar region, but he can hardly be considered an invalid, as he is able to follow his occupation as a shopkeeper with as much activity as could be expected of a man of his age and naturally feeble physique. His appetite is capricious, and his bowels are rather confined. The arterial tension is rather high, but not abnormally so; the vessels feel healthy, and there is no evidence of cardiac affection and no dropsy. There is marked anæmia; the red corpuscles number 1,750,000 per cubic millimetre; there is no poikilocytosis nor leucocytosis. Inoculations of the blood on agar, by my friend Dr. Warrington, show that it is sterile.

For some years he has had a small wart on the side of the nose near the inner canthus of the left eye. This was removed by my colleague Mr. W. Thelwall Thomas on November 16th, and proved to be a rodent cancer, which had not yet ulcerated. The patient made a rapid and complete recovery both from the chloroform and the operation.

The chief interest of the case lies in the remarkable condition of the urine. It is generally passed clear, when on inspection it presents nothing abnormal except that it is remarkably viscid and forms a very persistent froth. The quantity excreted in the twenty-four hours is not

much below normal. On three days when careful measurements were made it was 1660 c.c., 1000 c.c., and 1100 c.c. respectively, giving an average of 1220 c.c. (40 ounces).

The urine passed about the middle of the day between 11 a.m. and 5 p.m. is, however, as a rule turbid when voided, and looks at first not unlike milk, or a mixture of milk and urine, being white or nearly so. On standing it deposits an abundant white sediment, which may occupy as much as one sixth of the containing vessel. This sediment cannot be identified with any of the ordinary urinary deposits. Under the microscope it is seen to be amorphous, or more strictly to consist of minute structureless spherical particles. Collected on a filter, washed and dried, it forms a glue-like mass which gives the xanthoproteic and other colour reactions of a proteid, and is digested by an artificial gastric juice. It is almost insoluble in cold distilled water, and in dilute solutions of common salt, but is quickly dissolved in a cold weak solution of caustic soda (2 parts per 1000), in strong acetic acid, and to some extent in boiling water. When the deposit has been removed by filtration or decantation the remaining urine is for the most part identical with the clear urine which is passed at other times, and which I will now describe.

It generally contains a slight amount of mucus, and has the natural colour and odour of urine; it is generally acid, and its specific gravity is between 1015 and 1022. It is free from sugar and casts; the proportion of urea is about normal. It is remarkably viscid, almost syrupy in consistence, and forms a very persistent froth when shaken up with the air.

When carefully heated on a water-bath it begins to get turbid at about 50° C., sometimes at a lower temperature, and it becomes absolutely opaque between 56° and 60°, and a dense white precipitate begins to settle down. When this is removed by filtration, the filtrate gives no further coagulum when heated to the boiling-point, or at

most a faint cloud appears, showing that it is practically free from albumin.

The precipitate which forms at  $60^{\circ}$  is in all essential characters identical with the white sediment which is found in the turbid urine voided from time to time, and is manifestly the same body.

It is not properly a heat coagulum, as it is readily soluble in cold weak caustic soda (2 per 1000), and is partially redissolved if the urine is heated to boiling-point. It has, however, undergone some change during the process of heating to  $60^{\circ}$  C., in virtue of which it has lost the property of dissolving in urine at ordinary temperatures.

If a drop or two of dilute acetic acid be added to the urine before heating, the precipitation comes on at a much lower temperature, sometimes at  $31^{\circ}$  C. A very slight addition of acid will cause some precipitation to take place at the temperature of the body ( $37^{\circ}$  C.).

A portion of the urine was placed in a dialyser for some weeks. When it was heated without any addition a turbidity came on about  $60^{\circ}$  C., which could not be removed by filtering. Heated with a trace of acetic acid it remained almost clear, but if neutral salt was also present coagulation occurred.

The proteid body is also thrown down by mineral acids, sulphuric and hydrochloric as well as nitric, and is not to any notable degree soluble in excess of these reagents, unless by the aid of heat. The reaction with strong hydrochloric acid is distinct, even when the urine has been diluted with nineteen volumes of water, so that I look on it as a very delicate as well as convenient test for the body in question. It is best applied by the contact method, as in Heller's test for albumin. On the other hand, a large excess of acetic acid prevents its precipitation by heat, but it may in this case be precipitated by neutralisation, in a manner analogous to an albuminate.

When the urine is treated with concentrated nitric acid a precipitate takes place, which is partially dissolved by



heat and comes back on cooling, in the manner characteristic of an albumose. This reaction is not equally distinct in all the specimens of the urine, but is particularly well marked in the urine from which the salts have been removed by dialysis.

The body is also precipitated by saturation with ammonium sulphate and by absolute alcohol. The precipitate when fresh is dissolved by the simple addition of water, but after standing a few hours it is no longer soluble in neutral solutions, and so comes to resemble the precipitate formed by heat.

As regards the quantity of this body present in the urine, I find as the result of forty estimations made with Esbach's albuminometer, that the proportion varies from 6 to 16 per 1000. In one half the specimens it was over 10 parts per 1000. One specimen contained as much as 22 per 1000, which was confirmed by the gravimetric method.

The absolute amount on three days when the whole of the excretion was collected was 12·0, 10·5, and 12·9 grammes respectively. The largest proportion is generally found in the scanty urine passed in the middle of the day, but the absolute amount excreted seems pretty constant throughout the twenty-four hours.

The turbid specimens generally contain the largest proportion of proteid, but there are many exceptions to this. [See tables.]

The body which I have described as occurring in H—'s urine corresponds in most of its characters with a body which has been recognised as a constituent of the urine in about six cases during the last fifty years, and has been regarded for the most part as an albumose. It must not be confounded with the albumose which has been detected in minute quantities as a temporary constituent of the urine during the course of specific fevers and other diseases, and which presents very different characters from those which I have described. In the rare cases to which I refer it has occurred in large quantities and for prolonged

periods. The earliest known case of this kind is that investigated by Bence Jones, which will be found recorded in the 'Philosophical Transactions' of the Royal Society for 1848 (1). Since then no other case has been published in this country, with the possible exception of one to which I will presently refer, and which was not recognised as belonging to this class (5). The condition remained practically unknown until Kühne published a case in 1883, and since then five more cases have been investigated, which, with the present case, bring up the total to eight.

My case, however, appears to be unique in this respect, that a large quantity of the albumose is passed in the solid state, and in a form which could not fail to attract the notice of the most casual observer.

Bence Jones spoke of the body he found as a new substance occurring in the urine, and added some theoretical speculations as to its chemical constitution which are of no interest at the present time. It was precipitated from the urine by alcohol, and formed a coagulum with nitric acid, which dissolved on heating and returned on cooling. It was also precipitated by hydrochloric acid. Sometimes the urine yielded a coagulum on heating, but at other times it did not, although the reaction was acid. As it is not stated at what temperature coagulation took place it seems not unlikely that albumin may have been present at times, especially as casts were detected. In Kühne's case the urine behaved for the most part like H—'s, the chief difference being that the coagulum with heat and that with nitric acid were much more readily soluble on boiling in Kühne's case than in mine (2). Kühne concluded that the body was hetero-albumose, one of the products of the gastric digestion of albumin. The next case was reported by Huppert of Prague in 1889 (3). The reactions were identical with those in Kühne's case, and Huppert likewise looked on the body as hetero-albumose. A fourth case was recorded by Stokvis of Amsterdam in 1891, but I have not been able to ascer-

tain any particulars as to the behaviour of the urine (4). A remarkable case recorded by Dr. Byrom Bramwell and Dr. Noel Paton in 1892 is considered by Professor Huppert as belonging to the same category. The case was unique in this respect, that the proteid was easily obtained in the form of crystals (5). Dr. Bramwell described it as a crystalline globulin, and does not seem to have had any suspicion that it was in any way related to albumose, and I think we must hesitate before accepting Professor Huppert's conclusions as to its nature, in spite of the fact that its coagulation point corresponded to that of the body we are now considering.

A sixth case has been recently investigated by Matthes of Jena, under the direction of Professor Neumeister (6). Matthes's results in general correspond with those obtained by previous observers and by me, but he appears to have succeeded in obtaining the body in a state of greater purity than any of his predecessors have done. As the result of experiments with the purified substance he concludes that it is not hetero-albumose, and further, that it is not identical with any other known compound. Elementary analysis in his hands shows that it consists of carbon, hydrogen, nitrogen, sulphur, and oxygen only. Earlier observers found a considerable proportion of phosphorus, but Matthes believes this was derived from the nucleo-albumin which was also present. The process employed by Matthes to obtain the body in a state of purity has not yielded satisfactory results in my hands; and although by various methods I have obtained it in a solution practically free from salts, I am not satisfied that it is of sufficient purity to be submitted to elementary analysis.

A seventh case was under the care of Prof. Senator of Berlin in 1897, and has been recorded by Rosin (7).

As I have already pointed out, my case seems to be unique in this respect, that the albumose is often precipitated in the urinary passages, causing the urine to be turbid when it is passed, and I have tried to ascertain

the conditions which lead to this precipitation. I believe it is really of the same nature as the precipitation which takes place by heat, and that it is chiefly determined by an increase in the acidity of the urine. As I have shown, the addition of acetic acid to the urine causes coagulation to come on much below  $50^{\circ}$ , and even at a temperature below that of the body. The acidity of the turbid specimens is on the average considerably higher than that of the clear specimens (the exact figures are shown in the table), and the administration of an alkali to the patient causes the urine to remain clear for days together, although Esbach's test shows that the amount of the albumose present is as great as at other times. In Matthes's case the presence of salt was as necessary as an acid reaction to insure precipitation of the pure substance by heat; and, as I have stated, the dialysed urine in my own case did not coagulate when heated unless neutral salt was added. I therefore think the fact that the amount of turbidity is not always in proportion to the acidity may be accounted for by the modifying influence of the other constituents of the urine. The solubility at the boiling point of the precipitate formed with nitric acid also seems to be influenced by the amount of salts present, as it is most nearly complete when they have been removed by dialysis.

As regards the nature of the body my observations lead me to agree with Neumeister and Matthes that it is not hetero-albumose, but a peculiar body which is not identical with any other known compound. It differs from hetero-albumose in being soluble in distilled water and from the other digestion albumoses in being coagulated by heat.

As I pointed out in the beginning, there is nothing in the state of my patient to indicate the pathology of the condition, but I think the history of the other cases I have alluded to may lead us to a provisional diagnosis.

In Bence Jones's, Kühne's, and Huppert's cases there was some form of softening of bone; and in Stokvis's,

although there was no sign of bone affection during life, the autopsy showed that the bone marrow was replaced by a red gelatinous mass, the exact structure of which was unfortunately not ascertained microscopically.

In Byrom Bramwell's case the bones were not examined, but the kidneys were but slightly diseased.

In a private communication which I have received from Dr. Matthes he tells me that his case has come to an autopsy, and that the condition was that which has been described as multiple myeloid sarcoma; and in Prof. Senator's case the autopsy showed that several ribs were affected with a new formation, which was described as myelogenous round-celled sarcoma, but which had given rise to no objective symptoms during life.

I shall anxiously watch for any signs of affections of the bones in my patient, and I hope that the further progress of the case will form the subject of a future communication.

I have to express my best thanks to my friend Dr. O'Flaherty of Liverpool, who first sent me a specimen of the urine for analysis, and afterwards kindly placed the case at my disposal for purposes of investigation. I also wish to thank Dr. Lauder Brunton for the kindly interest he has shown in the case, and Professor Huppert and Professor Matthes for sending me copies of their various writings, to which I was unable to obtain access.

*Postscript*, April 9th, 1898.—Towards the end of December the patient became confined to bed in consequence of increasing weakness and distressing pains in the back and loins, which were much aggravated by movement. There was also marked tenderness over the ribs and sternum, especially at one spot on the latter at the level of the fourth intercostal space. In the beginning of February there was a good deal of bronchitis with viscid expectoration, which gave the nitric acid reaction for albumose. On the 5th, when sitting up in bed, a spontaneous separation of the second right rib from

the cartilage apparently occurred, followed by a localised painful swelling. Distressing dyspnœa and cough with rust-coloured sputum came on, and signs of pneumonia appeared at the left base. Latterly he has quite got rid of his lung troubles, has less pain, and is able to get up. There is, however, marked kyphosis in the dorsal region, with prominence of the dorsal spines from the sixth to the twelfth, and lordosis in the lumbar region, together with complete immobility of the vertebral column below the sixth dorsal vertebra. His friends think that he is shorter than he was before his illness, and his height is two inches less than he thinks it used to be. There are tender spots over the ribs and sternum. The main characters of the urine have not altered, but it is now seldom turbid. After it had been clear for a month a little benzoate of ammonia was administered, whereupon the acidity increased, and a dense turbidity appeared, which ceased when the drug was discontinued. On one or two occasions a few ill-defined granular casts were seen. The symptoms lead me to believe that the patient is suffering from some affection of the bones, probably the same as has been found in other cases of albumosuria.

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TABLE.

Time.	Amount in c.c.	Condition.	Sp. gr.	Acidity per mill. as oxalic acid.	Proteids Per 1000.	Proteids Amount in grammes.	Remarks.
Nov. 7th.							
A.m. 12.15	128	Clear	1019	1.449	12.0	1.536	Patient took about 2 grammes of Potass. Acetas during the day and on the three previous days. Clear during the four days.
" 4.50	312	"	1011	0.756	7.0	2.184	
" 8.30	313	"	1011	0.567	5.6	1.752	
P.m. 12.0	152	"	1014	0.504	5.8	0.881	
" 2.10	265	"	1018	1.008	9.0	2.385	
" 5.0	246	"	1017	0.630	6.0	1.476	
" 9.50	243	"	1017	1.134	8.0	1.944	
	1659					12.158	

Nov. 8th.—Potass. Acetas stopped. About noon a turbid specimen passed. Acidity 3.465 per mill., proteid 14.5 per mill.

Nov. 10th							
A.m. 4.30	310	Clear	1011	1.134	6.0	1.860	Patient took .5 c.c. Acid. Hydro- chloric. dil. three times a day after meals.
" 7.0	260	"	1013	0.964	6.0	1.560	
P.m. 12.30	80	Turbid	1023	3.276	16.0	1.280	
" 4.30	140	"	1023	2.502	12.0	1.680	
" 9.30	200	Clear	1020	2.835	12.0	2.400	
	990					8.740	



Nov. 18th	90	—	2·016	16·0	1·440	Patient took .5 gramme Ammon. Benzozas three times a day. The early morning's urine was pro- bably lost.
A.m. 10.0	85	—	3·150	15·0	1·275	
" 12.0	65	1020	2·772	13·0	0·845	
P.m. 2.0	84	—	2·502	12·0	1·008	
" 4.0	80	—	2·142	12·0	0·960	
" 5.30	335	—	1·134	8·0	2·684	
" 7.30	200	1019	1·953	14·0	2·800	
" 11.30		1022				
	939				11·012	

[illegible]

On an average the acidity of the turbid urine is greater than of that which is clear. Seven clear specimens which contain 10 parts per 1000 and upwards of proteid have an average acidity of 2'039 per 1000. Seven turbid specimens taken at random, but all containing not less than 10 parts per 1000, have an average acidity of 2'590 per 1000.

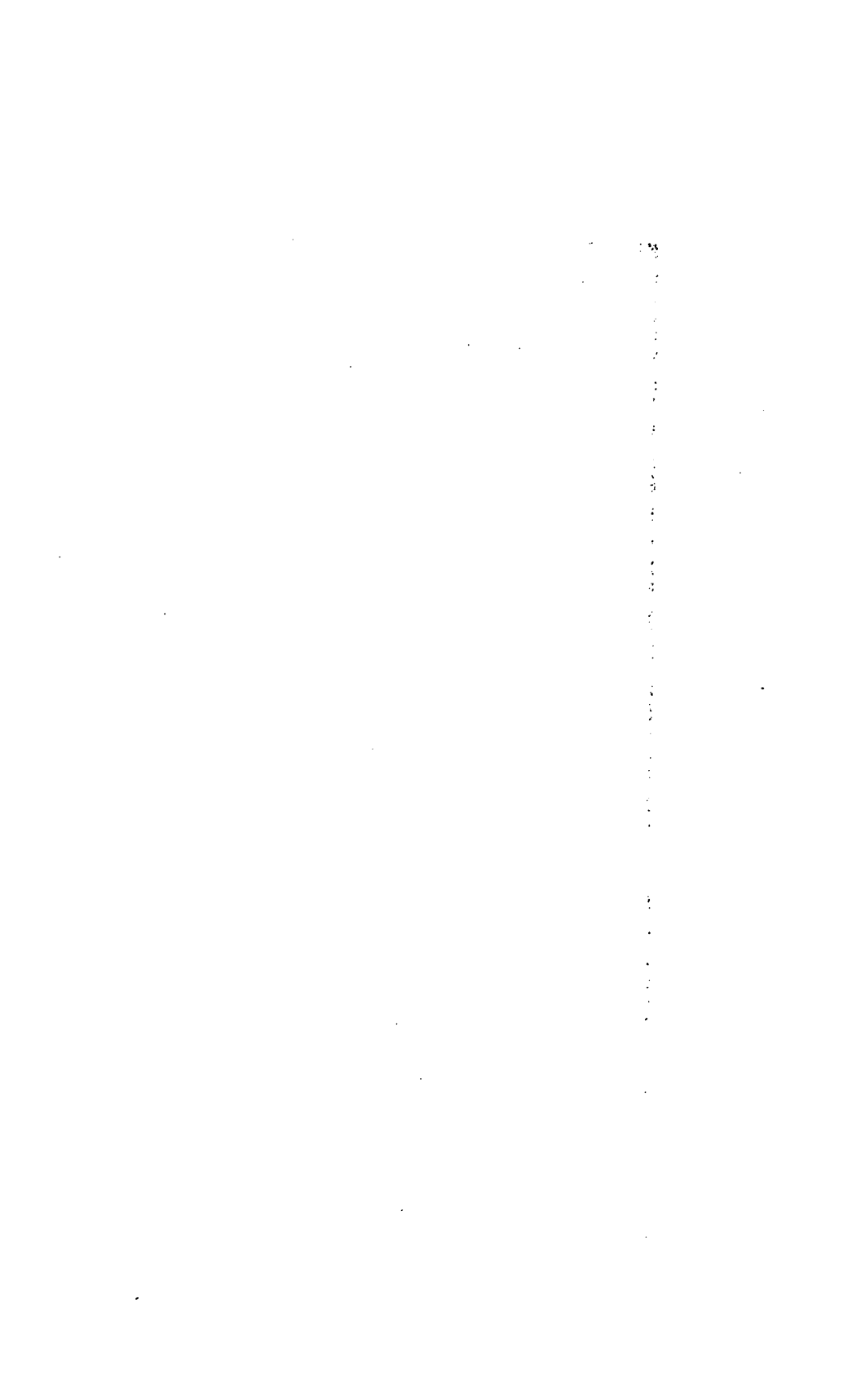
(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society, Third Series, vol. x, p. 119.)

## DESCRIPTION OF PLATE VII.

### A Case of Albumosuria (T. R. BEADSHAW).

The plate is reproduced from a photograph kindly taken for me in April, 1898, by Mr. Prosper H. Marsden, Dispenser to the Liverpool Royal Infirmary. The prominence in the dorsal region and the forward projection of the neck due to the kyphosis are obvious. It will be noticed that the integuments over the front of the thorax are thrown into transverse wrinkles. The lordosis in the lumbar region is indicated by the prominence of the abdomen.





# INTRA-PLEURAL TENSION

BY

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Received December 20th, 1897—Read May 10th, 1898

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UNDER normal conditions the lungs within the chest are in a constant state of tension. They are on the stretch and tend to contract by virtue of their own elasticity. Thus the two layers of the pleura, which in health are in close contact, are subject to a constant strain which tends to pull them apart.

This is called the intra-pleural tension. It is for all practical purposes equal in amount to the elastic contractility of the lung, but opposite in direction, and thus the elastic tension of the lung is positive but the pleural tension negative.

Whether, under normal conditions, where the two layers of the pleura are in close apposition, there is a force existing between them (viz. that of cohesion, as I suggested some years ago) which balances the elastic contractility of the lung or not, is a question which admits of discussion. *A priori* it is not improbable, and I have shown by experiment that it is possible. However

this may be, as soon as the two layers of the pleura are separated, cohesion, if it existed as an active force when they were in close apposition, becomes eliminated and the lungs tend to collapse.

The factors which combine to produce the intra-pleural tension are, first and foremost, the elasticity of the lungs ; secondly, the resistance of the chest walls.

These two factors, though varying considerably no doubt in different individuals, or even in the same individual under different conditions, are constant in any given individual under the ordinary conditions, and are much the same after death as during life.

Besides these there is a third factor, but it is one which is constantly varying, viz. the movements of respiration ; for it is evident that on deep inspiration the lungs are more on the stretch and the pleural tension therefore greater, while on expiration the lungs are less on the stretch and the intra-pleural tension therefore smaller.

If the air were stationary in the air tubes, as it is after death, the pressure in them would be that of the atmosphere ; but the movements of the air in and out during inspiration and expiration respectively, cause slight variations in pressure. Thus Landois and Stirling state that during inspiration the pressure in the lungs falls by a millimetre of mercury, *i. e.* half an inch of water, and during expiration rises by 1, 2 or 3 millimetres of mercury, *i. e.*  $\frac{1}{2}$  to  $1\frac{1}{2}$  inches of water. Thus the respiratory oscillation during ordinary respiration would be from  $1\frac{1}{2}$  to 2 inches of water.

The condition of the tubes, *i. e.* whether there be obstruction in them or not, is very important to bear in mind in determining either the elasticity of the lung or the pressure within the pleura ; for wherever there is any obstruction in them the elasticity of the lung will be *pro tanto* prevented from coming into play and the true results be not obtained.

If the forces of respiration be eliminated, as after death, then under the normal conditions the intra-pleural tension must be a negative quality, *i. e.* less than the atmospheric pressure, and less by as much as is represented by the contractility of the lungs.

During respiration also, so long as the breathing is quiet, *i. e.* not forced or violent, the intra-pleural tension must still be, both on inspiration and expiration, a negative quantity; for, assuming the value of the elasticity of the lung to be that which Donders estimated, *viz.* 6 to 7 millimetres of mercury, and that the respiratory oscillations are, as stated above, from 1 to 2 or 3 millimetres of mercury, there remain still 3 or 4 millimetres of mercury below which the tension will not fall.

Where the respirations are forced, that is to say where inspiration is deep or expiration excessive, the conditions may possibly vary considerably. With deep inspiration it must, however, always be negative, and the larger in amount the deeper the inspiration is.

With forced expiration, as in straining, coughing or vomiting, the pressure may be raised even to the extent of 3 or 4 inches of mercury (*i. e.* 75 to 100 millimetres), but as long as the two layers of the pleura are in close contact with the chest wall this excessive pressure is supported by the chest walls and does not fall directly on the pleura. On the other hand, under pathological conditions, where the two layers of the pleura are separated, any changes of pressure of the air within the lung are at once transmitted to the contents of the pleura; but the effect varies greatly, according as the contents of the pleura are practically incompressible as in the case of fluid, or easily compressible as in the case of air.

The methods by which the intra-pleural tension has been estimated in health are two:

1. By means of a trocar introduced into the pleura through the chest walls.
2. By means of a tube fastened into the trachea, the

pleural cavity being then opened by an incision through the chest walls, first on the one side and then on the other.

In either case the reading is made upon a mercury or water manometer connected with the trocar or the tracheal tube respectively.

In the second method, as the elastic tension in the lung is equal in amount to the tension in the pleura, but opposite in direction, the sign must be changed. Thus the positive readings showing the tension of the lung become negative readings in respect of the tension in the pleura.

For man both these methods of investigation are available after death, but the former only during life or under pathological conditions.

Intra-pleural tension is sometimes spoken of as intra-pleural pressure, and under pathological conditions the pressure is sometimes positive and sometimes negative. A certain amount of confusion is thus introduced both in thought and in expression.

This confusion will be removed if it be remembered that the records are expressed in manometer readings; thus, if the column of liquid in the manometer tube on connecting it with the pleura remains stationary, the reading will be zero, although in reality, of course, the actual pressure within the pleura is that of the atmosphere, and being equal to that within the air tubes might be called one; but as this is the zero point upon the manometer, it is more conveniently called "zero."

One, two, three, &c., millimetres of mercury or inches of water mean one, two, or three above or below the atmospheric pressure, as the case may be, and would be indicated by its appropriate sign of "plus" or "minus."

The readings are given in different investigations, sometimes in millimetres of mercury, sometimes in millimetres or inches of water. It is more convenient to use water, because the variations are greater, and therefore more obvious, and in the experiments which I have to



record I have measured it in inches ; but the conversion is easily made from one scale to the other ; thus one millimetre of mercury is approximately equal to half an inch or  $12\frac{1}{2}$  millimetres of water, and one inch of water is equal to 25 millimetres of water.

The intra-pleural tension as estimated by Donders was equal to 7 or 8 millimetres of mercury, and this is the value usually given in text-books.

Perl's experiments, conducted according to the second method, give a somewhat lower value, about 6 millimetres of mercury ('*Deutsches Archiv f. klin. Med.*,' 1869, vi, 1—5).

As Perl's experiments are the most interesting and important from our point of view, it seems to me desirable to give a short account of the results obtained by him, but in doing this I shall add various comments of my own.

Perl's experiments were conducted with a view to determine the pressure relations within the thorax in different diseases.

They were conducted in the following way :

A tube was fixed air-tight into the trachea, connected with a manometer ; the right pleura was then laid freely open and the pressure read ; after that the left pleura was laid open, and the pressure read again.

The observations were made upon the dead body of man.

1. Where the lungs are healthy the opening of one side of the chest goes a long way to satisfy the elasticity of both lungs, as the following figures show :

Pressure on opening the right side	.	29	...	49	...	39	...	32
"        "        "        left        "	.	11	...	5	...	9	...	7
		<hr/>		<hr/>		<hr/>		<hr/>
Total pressure (right + left)	.	40		54		48		39

This is very interesting, as it explains what we often observe in pneumothorax, viz. that the symptoms are the more severe the more healthy the patient has hitherto

been ; for if we take the total elasticity in the adult man, according to these experiments, to be on the average 50, the opening of the right side diminishes this elasticity by 40, leaving only 10 for the other ; and therefore the respiratory capacity of such an individual would be reduced by the opening of one side to the extent of four fifths—a difference which is not met with to the same amount where the lung is diseased.

2. Where the pleura is adherent the lungs are held back, and the recoil thus prevented, more or less, and sometimes entirely.

If the pleura be universally adherent, the contractility of that lung (or of both, if both be affected in the same way) will be entirely abolished ; for even if the elasticity be retained (and it seems to be retained sometimes), it will be prevented from acting. The degree to which the elasticity of the lung will be impaired depends upon the extent of the adhesions, and upon the secondary changes which they have produced in the lung tissue itself.

Where the pleura is partially adherent the opening of the remaining portion of the pleura on the one side does not produce the same marked effect upon the total elasticity of the lung as in the case of healthy persons.

If one pleural cavity be completely obliterated, the adhesions being universal, and the side be greatly contracted, the elasticity of the lung on the opposite side may be greatly reduced ; but, on the other hand, if the lung has undergone compensatory development, the elasticity may not only be not reduced, but may be actually increased. This is a very interesting and important observation, because it shows that the condition which is often described as “compensatory emphysema” is really a compensatory hypertrophy, as indeed there are the best clinical grounds for believing.

Thus, in a case of the kind, on the right side, where the pleura was completely adherent, the elasticity of that lung was reduced to 12 ; whilst on the opposite side (the left), where the pleura was free, although there was some

slight chronic tubercular change in the apex, the elasticity of the lung was 57.

The contractility of the lung is in all probability not purely elastic, that is to say it does not depend solely upon the elastic tissues in the lung, but probably also upon the muscular tissue with which the lung is so richly provided. Indeed, we may in this respect compare the lungs with the arteries and speak of "pulmonary tone" as we do of "arterial tone," and connect the tone in the lungs, as we do in the arteries, with the condition of the muscles, and possibly of the nerves.

If there be such a thing, therefore, as pulmonary tone, we might expect it to vary, as the tone of the vessels does, with the conditions of general health or of local disease, and there are some among Perl's observations which go far to prove this contention. Thus, without any local disease, the elasticity of the lungs was found greatly reduced in several cases of typhoid, in a case of severe hæmorrhage after ovariectomy, and in cases of delirium tremens, erysipelas, and phosphorus poisoning.

The condition would then be brought into close clinical association with that of acute tympanites, and like it would be a neuro-paralytic phenomenon, and of grave significance. For my own part, I believe there are good clinical grounds for believing that there is a neuro-paralytic condition of the lungs, strictly analogous with that condition of the abdomen called tympanites.

The two conditions might, of course, theoretically be associated, but when the abdomen is distended from any cause, whether it be from gas, effusion, or tumour, the displacement of the diaphragm upwards is so great that the tympanitic percussion obtained over the lungs may be due to mechanical rather than physiological or pathological causes.

Where the lungs are diseased, the other non-diseased parts may yield tympanitic resonance on percussion. This is a condition with which we have been long familiar

under the name of Skodaic resonance. There are several conditions under which this phenomenon appears. The commonest and easiest to explain is that in which it is associated with an effusion into the pleura. In this case, as the fluid forms the lung collapses and floats on the surface of the fluid, and over this collapsed lung, tympanitic percussion note is obtained. Its occurrence has been rightly referred to the relaxation of the lung tissue, and properly compared with the percussion note which is obtained over the lung removed from the body.

Again, conditions of complementary emphysema, where one part of the lung is collapsed and the other proportionately distended, may give rise to similar hyper-resonance.

But besides these there is, I believe, a third condition, and this requires a different explanation.

Nothing is much commoner with pneumonia than to find the parts of the lung above or in front of the affected portion yielding a highly tympanitic note, yet the pneumonic portions of the lung are certainly not collapsed or smaller than they should be, nor are they much larger; so neither of the explanations given is adequate, for where the hyper-resonance is obtained the lung is not collapsed on the one hand nor over-distended on the other. This condition, it appears to me, can only be explained on the assumption of a loss of lung-tonus, of neuro-paralytic origin, dependent on nutritive disturbance. This view is supported by some of Perl's observations; for example, there are several cases of pneumonia and some of embolism and gangrene, and in all of them the elasticity of the lung was very greatly reduced.

Acute miliary tuberculosis provides perhaps a still more striking instance of this condition, for the hyper-resonant or tympanitic note on percussion may be almost the only physical sign of disease yielded by the lung, and there can be little risk of error in assuming that the nutrition of the lung is profoundly affected in this disease.

In acute bronchitis, again, both in the child and in the adult, this nutritive disturbance in the lung probably explains in great part the tympanitic percussion obtained; but proof is more difficult in these cases, because of the other conditions which may take their share in producing it, viz. the collapse of some parts of the lungs and the complementary emphysema of others.

In estimating the intra-pleural tension and the elasticity of the lung the condition of the abdomen cannot be disregarded. During life there is the respiratory action of the abdominal muscles to reckon with, and after death we have to consider the effects of rigor mortis in them.

It is possible that the elasticity of the lung diminishes after death, but there are no direct observations which prove this, though it may be that this is the explanation of the difference as between Perl and Donders in their estimate of the average value of the elasticity of the lung.

Under pathological conditions the two layers of the pleura may be separated either by air or by fluid, and these present each of them its own peculiarities; thus fluid has weight, but it is practically incompressible; while air is compressible, but its weight may be disregarded. With fluid, therefore, the height of the column of fluid above the point of the trocar would effect the manometer reading, while with air the position of the puncture is indifferent.

With air the difficulties of the problem are in many respects less, and it will be well to begin with that condition first.

#### **Intra-pleural Pressure in Pneumothorax.**

The air may have gained access to the pleura either from without through the chest walls, or from within from the lung or some other air-containing viscus.

I. Where there is an opening through the chest walls.

(a) A punctured wound, such as is made by a stab with a penknife where the lung is injured as well as the external walls, we need not consider, for in this case, though the lung is wounded, air does not make its way in most cases into the pleura, but crosses the pleura and reaches the subcutaneous tissue. The explanation of this is very difficult, but need not be considered here.

(b) Where the opening through the chest walls is a large one, say at least as large as the section of the trachea, the air will pass freely in and out during respiration without let or hindrance as easily as it does through the trachea. In this case the pressure on the two sides of the lung, viz. that in the trachea and that in the pleura, will be equal always throughout all phases of respiration. Then the elasticity of the lung is free to come into play, and the lung collapses.

No doubt, as Donders said, the elasticity of the lung itself is able in time to produce complete collapse, but that this is not the result during life, daily observation demonstrates in cases in which the pleura is laid open by operation. In many of these cases where the opening is free, the lung, instead of being found completely collapsed, as might be expected, is on the contrary found distended to a certain point, and reaching, it may be, within an inch or so of the walls of the thorax; or if it has been collapsed, as for instance by an empyema, as soon as this has been evacuated the lung often rapidly expands so as to occupy the position just indicated.

Although it is true, when the respiratory movements are absent, as they are in the dead body, that the atmospheric pressure being equal on the two sides the elasticity of the lung will lead to its complete collapse, this is not true while the respiratory movements are acting, for the air does experience some obstruction in the air tubes, both on entrance and on exit; thus the pressure of the air in the air tubes during inspiration, is about half a millimetre of mercury ( $\frac{1}{4}$  inch of water) below that of

the atmosphere ; while on expiration it is as much as 2 to 3 millimetres of mercury (1 to  $1\frac{1}{2}$  inches of water) above it, and these differences are still greater if the respirations are violent and forced.

Under these circumstances it is clear that the lungs would only collapse until the pressure within them was equal to 2 or 3 millimetres of mercury, and that they would then cease to contract any more. If the lungs can be completely expanded under a pressure of 6 millimetres of mercury they could certainly be not less than half expanded, at any rate, under a pressure of 3 millimetres. What the exact relative volume of the lung might be actually under a pressure of 3 millimetres of mercury there are no observations to show.

When the expirations are forced, as in coughing, straining, or vomiting, the expiratory pressure becomes much higher than 3 millimetres of mercury, and the lung may then, if the external opening be free, be forced out again into actual contact with the chest walls or even made to protrude through the opening in the side.

Actual protrusion of the lung during coughing in these circumstances, however, rarely occurs, but the movements of the lung may be seen to be very free.

(c) Where the external opening is not large enough to allow the air to pass freely out during expiration, the excess of air which cannot escape is compressed, and the pressure rises above that of the atmosphere ; the lung is in consequence squeezed, and is more and more compressed until it becomes completely collapsed. The condition is then much the same as that in which the air has gained access to the pleura from the lung.

## II. Where the opening into the pleura is from within.

Theoretically there might be a condition in which the opening into the lung was large enough to permit the air to pass freely in and out on respiration without obstruction. This, however, as a matter of fact, hardly ever occurs. Air practically always enters the pleural cavity

more easily on inspiration than it finds exit from it during expiration.

The result is, as in the former case, that the lung becomes compressed during expiration, and the more rapidly so the greater the difference between the pressures of inspiration and expiration.

The division of pneumothorax into open, closed, and valvular thus becomes of little value from the present point of view, for all pneumothorax is at first more or less valvular, *i. e.* the air enters more easily during inspiration than it finds exit during expiration, and when the lung is completely collapsed, the pneumothorax practically becomes closed, whether the aperture be sealed permanently or not.

In pneumothorax the pressure conditions during inspiration and expiration require to be considered separately.

1. *The inspiratory pressure.*—When pneumothorax has occurred air will continue to enter on inspiration until the pressure within the pleura is equal to that of the atmosphere; as soon as this point is reached no more air can enter; consequently with a simple pneumothorax the inspiratory pressure cannot exceed that of the atmosphere, *i. e.* the reading on the manometer will be zero.

This is subject to one condition. In the early stages of pneumothorax the respirations are excessive and air will enter the pleura until the pleural pressure is equal to that of the atmosphere on deep inspiration. When after a time the urgent dyspnoea has passed off, so that inspiration is not so deep, the pressure may be above that of the atmosphere, to the extent of the difference between deep inspiration and ordinary inspiration; probably to a great extent this relieves itself in time by the absorption of a sufficient amount of the air to remove the difference.

If the inspiratory pressure is permanently above that of the atmosphere, that is to say is positive, there is required some other factor to cause the rise, and this, as a matter



of fact, we find to be the development of effusion, so that, speaking generally, a positive pressure in pneumothorax during inspiration indicates the presence of fluid.

2. *The expiratory pressure.*—Except in the theoretical and rare conditions in which there is no obstruction to the exit of air from the pleural cavity during expiration, the expiratory pressure in pneumothorax will always be positive in the early stages.

In these cases the heart and mediastinum are found displaced to their maximum and the lung completely collapsed.

The collapse of the lung, though capable of being brought about in time by its own elasticity, is always in chief part due to the compression exercised by the rise of expiratory pressure.

In the same way the displacement of the mediastinum, though capable of being brought about by the elastic traction of the opposite lung alone, is assisted by the raised expiratory pressure and caused to develop more rapidly or to be more extreme.

*The respiratory oscillation.*—The respiratory oscillation is the difference between the pressure on inspiration and the pressure on expiration, and this presents also some interesting points for consideration.

If the movements were violent the respiratory oscillations would, it might be thought, be considerable; but, as a rule this is not so, for on the affected side the chest is in the position of maximum inspiratory distension, so that no further expansion on inspiration is possible; while on the opposite side, owing to the displacement of the heart and mediastinum, the elasticity of the sound lung is reduced, so that the respiratory excursion is diminished, the result being that even with considerable dyspnoea the respiratory oscillation is not only not raised above the normal but may be considerably below it. It is, indeed, upon the defective respiratory excursion of the two sides that much of the dyspnoea depends.

Yet there are cases in which extensive respiratory

oscillations are seen, and it is important to consider the interpretation which may be given of them.

The number of cases of pneumothorax in which I have estimated the pressure in the pleura is eleven. In several of these paracentesis was performed more than once and the pressure determined, so that I have returns of the pressure in twenty paracenteses. In most of them I am able to record the inspiratory pressure, the expiratory pressure, and the difference between them, *i. e.* the respiratory oscillation.

In two cases the inspiratory pressure was that of the atmosphere, *i. e.* the manometer reading stood at zero at the time of the operation. In both these cases fluid was present as well as air, and from this it is evident that as the fluid formed the air must have been absorbed, since the opening in the lung in both cases was closed.

In another case the inspiratory pressure, after having been in the first two paracenteses positive, in the last two fell to zero, and in this case the change in pressure was due to the opening in the lung having become patent and being of considerable size.

In all the other cases the inspiratory pressure was positive, and fluid was present as well as air,—sometimes pus, sometimes serum. Although, as I have already stated, the inspiratory pressure in the early stages of pneumothorax may be positive to some extent, it can never be high, and practically we may conclude that when the inspiratory pressure is much raised, fluid is present as well as air in the pleura. This was at any rate the fact in the instances recorded.

The range of pressures in the different cases is considerable—thus the inspiratory pressure varied from 0 to 9 inches, the actual pressures being  $0\frac{1}{2}$ , 1,  $1\frac{1}{2}$ ,  $2\frac{1}{2}$ , 4,  $4\frac{1}{2}$ , 5,  $6\frac{1}{2}$ ,  $6\frac{3}{4}$ ,  $6\frac{7}{8}$ , 7,  $8\frac{1}{4}$ , 9; the expiratory pressure varied from 0 to  $13\frac{1}{4}$ , the respiratory oscillation from 0 to  $6\frac{3}{8}$ .

The amount of fluid removed is no indication of the amount of fluid present, for the effusion lies in the

diaphragm as it were in a saucer, and unless the point of the trocar be carefully manipulated so as to be beneath the level of the fluid, air will escape, the pressure fall, and no fluid be removed unless suction be used.

Suction, and especially the use of the aspirator, is in all cases of pneumothorax risky, for where the hole is patent it is useless except for the purposes of removing fluid, and then the trocar must be carefully manipulated; and if the opening be closed, suction is very likely to tear it open afresh.

The aspirator, therefore, is a dangerous instrument in pneumothorax, and should be rarely employed.

The ordinary syphonage apparatus, if properly manipulated, is all that is required and is devoid of risk.

It is not to be wondered at that the inspiratory pressure rises when fluid forms in pneumothorax, but it is surprising that the pressure is not much higher than it is. The highest pressure that I have to record is seven inches of water; and a pressure as high, or even higher, is met with in simple serous effusions.

It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms.

That the absorption of air from the pleural cavity is very rapid we know from experiments on animals and also from observations made in cases of accident and operation in man.

There are cases of pneumothorax even in man, in which, in spite of the presence of fluid, the inspiratory pressure is not above that of the atmosphere, as in the two cases mentioned.

There are two conditions under which the pressure might rise rapidly in an ordinary pneumothorax:

- (a) Where the effusion is poured out rapidly; the fluid must then be serous in character, so that time enough has not been given for the air to be sufficiently absorbed.
- (b) Where the lung or pleura is so far affected that

the absorption of air does not take place as readily as it should, and this no doubt is the fact in most cases of pneumothorax, and I think we shall not be going too far if in cases of pneumothorax of long duration we accept as an indication of somewhat extensive disease of both lung and pleura, the existence of a high intra-pleural pressure.

In two cases the pressure was determined at the end of the paracentesis as well as at the commencement of it. In the one case no suction was employed and the pressure fell to that of the atmosphere, the manometer registering zero. In another case some suction was used, and at the end of the operation the pressure registered — 9 inches of water. In another case the pressure, which on the first two paracenteses had been positive, in the third and fourth proved to be zero. The aspirator was then used, nothing but air was removed, and as this had no effect upon the pressure it was evident that the air passed freely into the pleura from the lung; in other words, that the opening was patent. This proved to be the case on the autopsy.

Further comment will be most usefully made in connection with the description of each case.

No. of case.	Sex.	Age.	Side.	I. Pr.	E. Pr.	R. O.	Amount of fluid removed.	Remarks.
1	M.	31	R.	0	0	0	oz. 80, turbid serum	11 weeks' duration; ultimately recovered.
2	M.	31	L.	0	1½	1½	11, pus	3 weeks' duration; marked phthisis.
				0	8	8	10	Great dyspnœa; 2nd paracentesis 7 weeks after the first; ultimate incision; necrosis of 6 inches of rib; death from exhaustion.
3	M.	30	L.	1½	2½	1	Pus	3 weeks' duration; at end of operation, Pr. = — 9; ultimately side discharged; incision; death.

No. of case.	Sex.	Age.	Side.	I. Pr.	E. Pr.	R. O.	Amount of fluid removed.	Remarks.
4	M.	24	L.	8½	—	—	oz. 34, pus 41, pus 7 8½ 1½ Much	3 weeks' duration. 14 days later. 3 days later; free opening formed in lung; incision subsequently; death.
5	M.	22	R.	6½	13½	6½	41, sero-pus 22, sero-pus 28, sero-pus 36	3 months' duration. 3 weeks later; ultimate recovery. 5 weeks' duration.
6	M.	21	R.	1	5	4	28, sero-pus 36	17 days later; subsequent incision; death from hæmoptysis. P.M. Ruptured pulmonary aneurysm.
7	M.	24	L.	4	5	1	22, serum 6, pus	3 days' duration; recovery.
8	M.	35	L.	6½	7	½	6, pus	Many months' duration. At end of operation I. Pr. = 0, R.O. = 2. Was pointing at time; therefore pointing pyopneumothorax may have low or even zero pressure. Interesting case to compare with empyema.
9	F.	23	L.	2½	9	6½	Several, pus	3 days' duration. Much dyspnoea at time of operation. At end of operation R.O. = 1, dyspnoea passed off.
				4½	5	½	—	3 days later, owing to little fluid being obtained, suction employed up to - 36 inches of water, but only 6 oz. more fluid obtained. ∴ Lung bound down. Incision. Death from shock immediately on incision. No p.m.
10	M.	46	—	9	9	0	74 Serum	10 days' duration. 16 days later, on deep Inspir. Pr. = - 1; on cough Exp. Pr. = 7. Ultimately puncture suppurated, though fluid was serous. Patient lived 12 months, and died of phthisis.
11	M.	22	L.	5	8½	3½	36, serum A few — 40, pus	A few days. 9 days later. Opening had become patent. On death large patent opening found in lung.

### Intra-pleural Pressure in Pleural Effusion.

1. *Serous effusion*.—In health the pleural cavity contains no fluid, and we often speak of it as dry; yet this is inaccurate, for there is in all probability a constant circulation of fluid into the pleura and out of it, the fluid being effused by the blood-vessels and carried away by the lymphatics. The mechanism by which this is performed has been described as the lymphatic pump. It consists of the lymphatic vessels with their stomata and valves, and is worked by the respiratory movements. The course of the circulation in the lung is from the pleural surface (the periphery, that is to say), towards the root of the lung, as has been determined by experiment, and there is a similar circulation from the pleural surface through the diaphragm and through the chest walls.

There are two ways, therefore, in which fluid may accumulate in the pleura; either it may be poured out into the pleura in larger quantities than the pump can remove, or the amount of fluid may not be above the normal but the pump be defective.

In the case of pleural inflammation both these processes probably come into play; the amount of transudation is considerable, and the stomata and smaller lymphatics are often plugged by a deposit of fibrin. Thus in inflammatory cases the fluid may accumulate with very great rapidity and soon reach a large amount.

In a case of dropsy of the pleura consequent, for instance, on heart disease, the explanation is probably also mechanical. Exudation under these conditions takes place from the blood-vessels into the lymphatics of the lung, which become water-logged or choked, and thus unable to carry off the fluid from the pleural cavity, so that it accumulates in it.

With dropsy, however, the development of fluid is much slower and the amount as a rule much less.

When fluid collects in the pleura it falls by its weight

to the lowest part, and although the tension in the whole pleural cavity will be diminished in proportion to the amount of fluid present, still the effect upon the different parts of the lung will be different; thus the lowest parts will suffer most and become collapsed as we know they do, while the upper parts of the lung remain distended; but the tension in the upper part of the pleural cavity is lower than it otherwise would be, as is shown by Calvert's observations ('St. Barthol. Hosp. Rep.,' 1892, p. 131).

This diminished tone in the lung or tension in the pleura explains the hyper-resonant note which is obtained in those parts of the lung which are floating upon the fluid.

In determining the intra-pleural pressure in cases of fluid effusion something will depend upon the seat of puncture, as Calvert also has shown, for if the mouth of the trocar be 1, 2, or 3 inches respectively below the level of the fluid, there will be the pressure of a column of fluid of this height to allow for.

If, for example, the intra-pleural tension be equivalent to 3 inches of water, and the amount of fluid exuded into the pleura be sufficient to reduce this 3 inches negative pressure to 2 inches negative pressure, it follows that if the mouth of the trocar be 2 inches below the level of the fluid, a positive pressure of 2 inches will have to be added to the negative pressure in the rest of the pleura, which will reduce the pressure-reading to zero; or if the height of the fluid be 3 inches instead of 2, would convert the pressure at the point of puncture to a positive pressure of 1 inch.

In most of my cases I have taken as far as possible the same relative position in the chest for puncture, viz. the middle of the axilla, as the patient is lying upon the back; the results are therefore more or less comparable with one another; but although I have endeavoured to make some allowance for these considerations, the readings in pleural effusions have not anything like the same value as those in pneumothorax.

I have determined the pressure in twenty-seven cases of serous effusion, some of which were tapped more than once, so that I have thirty-one pressure-records to found these observations upon.

In the table which follows I have arranged the cases in order according to the size of the effusion.

No. of case.	Sex.	Age.	Side.	Amount of fluid removed.	Pressure.	Resp. osc.	Remarks.
1	M.	46	R.	oz. 139	2½	0	At end of operation Pr. = -9½; resp. osc. = ½; spontaneous coagulation in fluid.
2	M.	41	—	130	18	$\frac{1}{15}$	
3	M.	32	L.	112 After 6 days 70 " " 72	-1 -1 +3½	3 1 1	
4	M.	39	L.	106	8	1½	After paracentesis fluid rapidly disappeared; Pr. after first paracentesis = -1.
				19	5	1½	
5	M.	35	L.	98	4	1	Fluid apparently formed in 5 days. Blood-stained fluid.
6	M.	39	—	85	6	½	
7	M.	51	R.	77	5	2	
8	Cf. Case	3		72	3½	1	
9	M.	42	L.	72	1½	1	
10	Cf. Case	3		70	-1	1	
11	M.	22	L.	68	8½	—	On second paracentesis Pr. = 0; resp. osc. = 8 (from +4 to -4); a few ounces of pus.
12	M.	33	R.	62	0	1½	
13	M.	54	L.	60	0	0	
14	M.	63	R.	55	4	2	Died shortly after of acute phthisis.
15	F.	25	R.	52	8½	1	
16	M.	34	R.	50	2½	—	
17	F.	29	L.	47	2	$\frac{3}{4}$	Sp. gr. of fluid 1040; spontaneous coagulation.
18	M.	83	L.	45	11	1½	
19	F.	46	L.	46	11	—	
20	M.	23	L.	40	0	$\frac{3}{4}$	Fluid blood-stained. 100 ounces removed previously, but pressure not taken.
				20	3	$\frac{3}{4}$	



No. of case.	Sex.	Age.	Side.	Amount of fluid removed.	Pressure.	Resp. osc.	Remarks.
21	F.	—	—	oz. 40	1½	¾	Sudden death. Pleurisy secondary to sarcoma of vertebræ.
22	—	—	—	34	0	2	
23	F.	53	L.	30	½	½	
24	Cf.	Case 19		20	3	¾	
25	Cf.	Case 4		19	5	1½	
				Amount not specified.			
26	F.	34	—	Very large	1	¾	At end of operation Pr. = + 3. Cast expectoration subsequently from trachea. Death.
27	M.	26	L.	Large	8	—	
28	M.	—	L.	Moderate	0	2	
29	M.	62	L.	Large	11½	—	
30	M.	46	L.	Small	0	—	
31	F.	19	—	Moderate	-4	½	

A glance at this table will show that the pressures vary greatly and irregularly, and that there is no definite relation between the size of the effusion and the amount of pressure; for there are among the cases instances of large effusions with low or even negative pressures, and of small effusions with high pressures.

Thus a large effusion was found :

With *negative* pressure in—

Case.	Amount.	Pressure.
3 ...	112	— 1
A few days later	70	— 1

With *low* pressure—

26 ...	large	1
1 ...	139	2½

With *high* pressure—

2 ...	130	18
4 ...	106	8
5 ...	98	4
6 ...	85	6
27 ...	large	8
29 ...	large	11½

In the same way with **moderate** effusions the pressure may be negative, zero, moderate, or high.

With *negative* pressure—

Case.		Amount.		Pressure.
10	...	70	...	- 1
With <i>zero</i> pressure—				
12	...	62	...	0
13	...	60	...	0
28	...	moderate	...	0
With <i>high</i> pressure—				
7	...	77	...	5
11	...	68	...	8½
15	...	52	...	8½
14	...	55	...	4

**Small effusions in the same way :**

With *zero* pressure—

20	...	40	...	0
22	...	34	...	0

With *low* pressure—

23	...	30	...	½
21	...	40	...	1¼

With *high* pressure—

18	...	45	...	11
19	...	46	...	11
24	...	20	...	3
25	...	19	...	5

If we look at these cases again from the point of view of pressure, we find :

**A negative pressure—**

With *large* effusion in—

3	...	112	...	- 1
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With *moderate* effusions in—

10	...	70	...	- 1
31	...	moderate	...	- 4

**A zero pressure—**

**With moderate effusions in—**

Case.		Amount.		Pressure.
12	...	62	...	0
13	...	60	...	0
28	...	moderate	...	0
30	...	small	...	0

**With small effusions in—**

20	...	40	...	0
22	...	34	...	0

**Very high pressures—**

**With large effusion in—**

2	...	130	...	18
29	...	large	...	11½
4	...	106	...	8

**With moderate effusions in—**

11	...	68	...	8½
15	...	52	...	8½

**With small effusions in—**

18	...	45	...	11
19	...	46	...	11

It might be thought that this irregularity could be explained by the different stages in which the inflammatory effusions were at the time of investigation; for instance, that where the inflammation was active the pressure might be high; where the inflammation was subsiding the pressure might be low; and that when the fluid was being absorbed the pressure might even become negative.

Case 6 might, perhaps, bear this interpretation, for the fluid in this case was accumulating with very great rapidity, and within four or five days from the very commencement of the illness 85 ounces of fluid were removed from the pleura; the pressure in that case was + 6; but this theory is not supported by many other cases, and I am afraid the whole matter is by no means so simple as this would make it.

Two cases present a special interest because the para-

centesis was repeated, and the pressure determined on subsequent occasions. Case 3 is an instance of the kind. On the first paracentesis 112 ounces were removed, and the pressure was  $-1$ . Six days later 70 ounces more were removed, and the pressure was  $-1$  again. Six days later still 72 ounces were removed, and then the pressure was  $+3\frac{1}{2}$ .

Case 19 is another instance of the kind. On the first paracentesis 100 ounces were removed, and the pressure was not determined. On the second 40 ounces were removed, and the pressure was zero. On the third 20 ounces were removed, and the pressure was  $+3$ .

It is tempting to suppose that by the third paracentesis in each of these cases the fluid had become encapsulated, the pleura being adherent elsewhere, and that this might account for the curiously sudden rise in pressure which occurred in both cases.

In three cases the pressure was determined at the end of the operation as well as at the commencement of it. In Case 1, where 139 ounces were removed, the pressure at first was  $2\frac{1}{2}$ , while at the end of the operation it was  $-9\frac{1}{2}$ . In Case 4 the pressure at the commencement of the operation was 8, and at the end of the operation  $-1$ ; and in Case 29, where the pressure was  $11\frac{1}{2}$  at the commencement, it still remained  $+3$  at the end.

From this analysis of these observations I think it is clear that the intra-pleural pressures in serous effusions are by no means so simple as they might be thought to be, and that the whole subject requires a great deal of further investigation.

#### *The Respiratory Oscillation.*

The respiratory oscillation is, as already stated, in health about 1 to  $1\frac{1}{2}$  inches of water, and in fifteen of the cases recorded the respiratory oscillation was of about this amount.

It was reduced to zero, that is there was no respiratory oscillation at all in three cases: Case 1 with 139 ounces of

fluid; Case 12 with 62 ounces of fluid; and Case 13 with 60 ounces of fluid.

It was also below 1 inch in nine cases. On the other hand, it was increased only in one, viz. Case 3, in which with 112 ounces of fluid and a negative pressure,  $-1$ , there was a respiratory oscillation of 3 inches.

The respiratory oscillation, therefore, is generally reduced in serous effusions, and may be completely absent.

Inasmuch as the action of the lymphatic pump depends upon the respiratory movements of which the respiratory oscillation is the index, it is evident that where the respiratory oscillation is absent the mechanism of the removal of fluid must be defective.

Where, with a large effusion at the time of introducing the trocar, the respiratory oscillations are seen to be absent, they frequently return after the fluid has been removed, and may even be above the normal. Even the partial removal of the fluid, *i. e.* the removal of a part of it and not the whole, may cause the respiratory oscillations to return. This explains what is frequently observed and is difficult otherwise to understand, viz. that the removal of a small quantity of fluid not infrequently leads to the rapid spontaneous disappearance of the rest; the interpretation is that the lymphatic pump has been started to work again.

Other points which these tables show are that pressures have no relation to age, for the cases range from the young to the old; nor to sex; nor to the side affected; nor is the pressure any help in prognosis, for nearly all the cases in this list recovered with the exception of three, one of which died of rapidly advancing phthisis, the second of cancer, and the third with sarcoma of the vertebræ.

These observations have a bearing also upon the mechanism of the displacement of the heart and mediastinum in pleuritic effusion, for they show that displacement does not necessarily depend in any way upon pressure, for the maximum displacement may be found with a

zero pressure or even with a negative pressure on the affected side.

We may conclude that the intra-pleural pressure in serous effusions is not as simple as it might *a priori* be thought to be; that it depends upon many factors, which are difficult to allow for, and that the whole subject requires further investigation.

### Intra-pleural Pressure in Empyema.

I have ten observations of the pressure in empyema. They show that the question of intra-pleural pressure in empyema is much simpler than in the case of serous effusion, and completely in accord with what we should expect from our knowledge of suppuration in other parts.

We know that in other places the formation of pus is attended with considerable tension,—in other words, that it goes on under considerable pressure. It is only in the very chronic, so-called cold abscesses that the tension is low; but even then the pressure is probably considerably above that of the atmosphere.

No. of case.	Sex.	Age.	Amount removed.	Pres. sure.	Resp. osc.	Remarks.
1	M.	34	oz. 91 56	16 10	0 0	Subsequently expectorated pus. Developed pneumothorax and died with phthisis. After whooping-cough.
2	M.	5	6	4	0	
3	F.	19	2	3	$\frac{1}{2}$	
4	M.	28	14 $\frac{1}{2}$	5 $\frac{1}{2}$	—	
5	M.	29	20	3	1	
6	M.	6 $\frac{1}{2}$	40	8	$\frac{1}{2}$	
7	M.	26	15	8	0	On subsequent paracentesis 15 ounces removed twice; incision and recovery.
8	F.	13	15	5	$\frac{3}{4}$	On subsequent paracentesis 6 ounces and 1 ounce removed; cure by paracentesis only.
9	M.	35	6	7	$\frac{1}{2}$	

It will be seen that among my observations in all cases the pressure is considerably increased. The lowest of the series is 3, and the highest 16.

The highest pressure is found, as it happens, with the largest effusion, for in this case 91 ounces were removed with a pressure of 16, and on a subsequent paracentesis 56 ounces were removed with a pressure of 10.

There is, however, no necessary relation between the size of the effusion and the height of the pressure; for if small empyemata are localised, as they frequently are, the pressure may be very high, though the amount of pus contained in them be very small.

There is one interesting case which bears upon this among the observations in serous effusion, where a serous effusion of some size was tapped, but the pressure not raised.

On the third paracentesis the pressure was found to be +3, but the effusion then was no longer serous but purulent, the conclusion being that the serous effusion had been followed by a small local empyema. This was incised and the patient recovered.

The respiratory oscillation in empyema also is interesting; in all cases it was reduced, and in many of them absolutely disappeared. In four cases it was zero, in one  $\frac{1}{4}$ , in two  $\frac{1}{2}$ , and only in two instances did it approach 1.

The two points, therefore, in reference to the pressures in empyema are, first, that it is always positive, and sometimes considerably raised; and secondly, that the respiratory oscillation is practically absent.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 130.)





# A CASE OF CYST OF THE URACHUS

WITH NOTES ON URACHAL AND SO-CALLED  
"ALLANTOIC CYSTS"

BY

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EARLY in the autumn of 1897 I operated on a case of cystic abdominal tumour which presented certain remarkable features. After much deliberation I am of opinion that the tumour arose from the urachus. Cysts attributed to that structure have been repeatedly described, and interesting monographs on the subject have been written by Wutz, Freer, Byron Robinson, and other writers, mostly German and American.

Before considering urachal cysts in general I will relate my own experience.

E. S—, aged 59, married thirty-one years (five children, two abortions, last confinement twenty-two years ago), a patient of Dr. Blamey, of Penryn, Cornwall, was admitted into the Samaritan Free Hospital on September 28th, 1897. The patient had enjoyed perfect health till three years previously, when she suffered from bronchitis

after influenza. Ever afterwards she was troubled with all the symptoms of emphysema. For about nine months she had been subject to another complication, pain in the lower part of the abdomen, chiefly towards the left iliac fossa. Two months before admission Dr. Blamey's attention was turned to an abdominal swelling of considerable size lying somewhat to the left of the middle line. At that time symptoms of peritonitis came on periodically, subsiding under rest and opium. The tumour became rather smaller.

The patient was somewhat thin, her cough troubled her, and emphysema was marked at both apices. A spherical swelling occupied the middle line of the abdomen from the umbilicus to the pubes. It bulged perhaps a little more to the left than to the right. At first it seemed solid although elastic, but fluctuation could be felt after the patient had rested for a few days. The swelling could apparently be moved laterally, but returned into its place when let go.

On vaginal exploration the cervix was found fairly long, lying backwards. The sound passed two inches, somewhat forwards. The uterus seemed to slide laterally behind the tumour.

The tongue was clean and moist, the appetite much impaired since the recent attacks of abdominal pain, and the bowels were sluggish. During the first week spent in the hospital about 40 ounces of urine were passed daily. It was always clear, and never contained albumen, but varied considerably in colour and in specific gravity (1009 to 1022) daily. During the same period the temperature ranged from  $97.6^{\circ}$  to  $98.8^{\circ}$ ; the pulse was 96, regular, small in volume, and hard.

Diagnosis was very uncertain. Ovarian cysts are so frequent, and vary so much in character, that I was inclined to take this swelling for a tumour of that familiar type. At the same time I bore in mind Dr. Blamey's history, and suspected that the swelling might be a circumscribed collection of peritoneal effusion. The latter

explanation seemed to be verified at the operation; but after due deliberation and consideration of notes, which I took directly after I closed the abdomen, I came to the conclusion that adhesive peritonitis had evidently taken place around the limits of the tumour, but that the tumour itself was not an encysted dropsy of the peritoneum.

On October 6th I operated, assisted by Mr. Corrie Keep. On dividing the aponeurotic layer of the wound, a thick, tough, yellow membrane was exposed. I incised it carefully; about a pint of a dirty greenish-brown fluid escaped, with shreds resembling products of inflammatory effusion. I washed out the cavity thus laid open with iodine water; then I opened it more freely, and found that close to the umbilical level of its wall the yellow membrane passed backwards, where it formed a well-defined septum between its cavity below and adherent intestine and omentum above. Beyond the region of adhesion the intestines seemed healthy. The attacks of peritonitis could now be accounted for.

I traced the yellow membrane downwards, and came upon a second and smaller cyst or loculus immediately below the larger cavity. The yellow membrane bounding the latter passed directly backwards over the top of the lower cyst. I have now no doubt that the same membrane formed the back of the upper cavity. I naturally suspected, from its position, that the lower cyst might be the bladder, as inferiorly it lay in connective tissue, and was closely applied to the parietes in front. A sound was passed and held in the bladder. When that instrument was pushed as high as possible I found that it was behind the cyst. I tapped the latter, and about half a pint of dirty greenish-brown fluid escaped. On opening this lower cystic cavity freely I could feel the tip of the sound behind it. The walls could not safely have been separated from the bladder. When mopped with absorbent gauze the interior of the cyst remained dry; no fluid resembling urine ever escaped afterwards from either the upper or lower cavity. I packed the lower cavity with

iodoform gauze, took pains to close the general peritoneal cavity near the umbilicus, so that it could not be infected, and lastly packed the greater cavity. Its inner wall posteriorly felt rough all over, and was fairly uniform; it was, in short, something more than mere effusion covering intestine, and was continuous with the reflections of the yellow membrane above described. I cut away the contiguous boundaries of the two cavities in order to facilitate packing and drainage. Two stout red rubber drainage-tubes were inserted into the lower angle of the wound.

On the third day the iodoform gauze was removed; it was blood-stained, as might be expected. Nothing but serum, ultimately clear, flowed from the tubes; they were therefore removed. For a fortnight the cavity was washed out with iodine water. During convalescence there was neither fever nor rise of pulse, the urine exhibited no morbid conditions, and there was no discharge of any kind of fluid from the cavity. No symptoms of peritonitis were observed, nor was there any trouble about flatus.

On October 27th the patient was discharged from the hospital. The uterus was fairly moveable, the fornices free, and there was no dysuria.

On November 11th I saw her again. The cavity was reduced to a small sinus; she had syringed it daily with a weak solution of carbolic acid. There was resonance all over and around the cicatrix, under which a hard area could be felt, probably the shrunken cyst wall. The uterus remained fairly though not freely moveable. Douglas's pouch and the lateral fornices were quite free.

March 6th.—Dr. Blamey wrote: "Mrs. S— is now quite well. The little wound was somewhat stubborn, but has now quite healed up."

A small piece of the wall of the larger cyst cavity was preserved, and Mr. Corrie Keep made sections of it. We fancied that there was tissue like muscular fibre in its substance. I therefore took a section to Mr. Shattock, who kindly made the following report:

" Royal College of Surgeons ;

" Jan. 26th, 1898.

" I have very carefully studied the section of (?) urachal cyst.

" Firstly, there is no histological sign of tuberculosis.

" On the convex under aspect the section shows a fairly broad zone of close fibrous tissue, but in the rest of its extent the preparation consists mainly of unstripped muscle-fibre arranged in bundles (Pl. VIII, fig. 1). I am quite satisfied that this is so, as one can see the cross-sections of the muscle-cells with small circular (in sectional area) nucleus lying in the cell body, which latter can, moreover, be traced for a considerable distance, by altering the focus, beyond the termination of the nucleus. In the longitudinal sections of the muscle-bundles here and there isolated cells of the typical elongated, tapering form, with central staff-shaped nucleus, are encountered.

" There is no epithelial lining and nothing like a mucosal corium, apparently from artificial damage, since the actual inner surface is constituted by frayed-out vascular tissue, and is not even granulating.

" I think, therefore, that the diagnosis of urachal cyst is securely established.

" S. G. SHATTOCK."

I will defer all comment on this case until after consideration of the somewhat similar experiences of other operators. Before turning to their reports, I think it advisable to dwell a little on the normal urachus itself and on the experience of pathologists, which at least proves that minute cystic dilatations of its canal must be frequent. After considering the records of suspected cases where an operation was performed, I will in conclusion turn the Society's attention to a remarkable type of abdominal cyst, originally described by Mr. Lawson Tait, who ascribes it theoretically to the allantois. His cases have been repeatedly quoted, and much that he himself rates only as theory has been accepted as fact. Hence

they are important enough to deserve full consideration, and obscure enough to demand the most critical scrutiny.

*The Normal Urachus.*

The homologies of the urachus are so well determined that I need say nothing about its comparative anatomy.

We are all familiar with this fibro-muscular cord which runs between the summit of the bladder and the umbilicus. Posteriorly its attachment to the bladder corresponds to the reflection of the parietal peritoneum on to that organ. Hence it marks the limits of the portion of the bladder invested by peritoneum, as immediately in front of the urachus and from thence forwards and downwards the bladder has no peritoneal covering, but is separated from the body of the pubes and the abdominal wall by connective tissue sufficiently loose to allow it to move as it fills and empties itself of its contents.

This connective tissue lies in front of the urachus itself, and usually is less abundant as the cord passes in front of the peritoneum upwards to the umbilicus; yet I have found the urachus lying behind very loose subserous connective tissue all along its track. In cases of abdomino-pelvic tumour this tissue is often very abundant, while the urachus undergoes hypertrophy.

Thus the cellular surroundings of the urachus must allow of the free expansion of any cyst, or any collection of fluid without a true wall, in their neighbourhood. Of course this applies to a true urachal cyst, but it does not follow that every cyst in this region is urachal. The connective tissue might suppurate from injury or anterior parametritis, and an abscess cavity might remain and assume a cyst-like character. Hydatids, too, must not be overlooked.

When chronic peritonitis exists, this connective tissue becomes soldered to the transversalis fascia and to thickened peritoneum, greatly confusing relations, especially if

an ovarian cyst with thick laminated walls happen to be adherent to the peritoneum. The urachus, thickened and otherwise altered, may be a stumbling-block instead of a guide. I have myself divided it, laying bare a patulous portion of its canal, in one of these complicated cases, and can understand that the discovery of a tubular structure on the front of an inflamed cyst might puzzle an unwary operator.

*The Canal of the Urachus.*

This leads to the question how far the canal of the urachus may be pervious in the adult. I believe that it is impossible to draw the line between the normal and the abnormal in this question. The origin of the urachus may look very like a diverticulum, as in one female subject in the Normal Anatomy Series of Dissections by Mr. Pearson at the Museum of the Royal College of Surgeons. In this case a small digital protrusion may be seen running for half an inch from the top of the bladder till it abruptly joins its smaller and more normal portion. In another subject in the same series there is a distinct indication of a diverticulum, whilst in another not the faintest trace of such a protrusion can be detected. In abdominal operations I have noted similar variations. Most probably more or less patency is the rule. Luschka (Pl. VIII, fig. 2) and Wutz's researches should be followed up; the latter observer, who examined seventy-four subjects, found that the epithelial part of the urachus steadily grows up to the twenty-fifth year, its canal becoming wider. Its growth is, however, less rapid than that of the tissues of the abdominal walls, so that it ceases further and further from the umbilicus as the subject grows older. Wutz distinguished a transverse valvular fold to the vesical orifice of the urachus which prevents the passage of urine into the urachal canal (Fig. 5). This seems to

explain why urachal fistula has been found to develop when there is obstruction to the passage of urine out of the urethra, and why urinary fistula sometimes develops after incision and drainage of a urachal cyst which at the operation seemed unconnected with the bladder. Wutz's valve is, in fact, liable to be forced open by the pressure of urine retained in the bladder. The canal of the urachus is lined with stratified epithelium below. Wutz distinguished three layers; higher up the layers are reduced to two, and near the upper extremity of the tube to one. He found that the cells individually vary in shape; some are flat, some large and oval with big nuclei, some bear one or more long processes. Much has been made of these statements, as will be seen; but before we feel certain about the significance of cells in the walls of cystic tumours in the abdominal parietes it would be better for us to investigate Wutz's researches or to entrust such an investigation to a competent histologist. Under the epithelium of the canal and its subepithelial connective tissue there is a layer of plain muscle-cells.

The muscular sheath of the tube, the middle true ligament of the bladder, becomes more and more mixed with white fibrous tissue the nearer the urachus approaches the umbilicus, so that at length it is reduced to a tendon (Pl. VIII, fig. 2). Mr. Keep has prepared for me the sections, which I now exhibit (Pl. IX, figs. 3 and 4), of the urachus of a woman aged 45. Plain muscle-cells are to be seen in abundance, arranged with much regularity in bundles which nearly all seem to run vertically. In the child the urachus remains muscular half the way up; in the adult the tendon begins lower down. It is clear that a pervious urachus is very frequent, if not the rule, in the human body.



*Urachal Fistula.*

Of all pathological conditions affecting the urachus, it is urachal fistula that is best known to the surgeon. It allows of the escape of urine through the umbilical orifice, and even calculi may come away through it. In the most aggravated form the urachus is represented by a long and wide process of the bladder opening at the umbilicus. Many cases have been published. A patulous condition of the lower part of the urachus may give trouble, though the canal remain closed near the umbilicus.

In the early days of the Pathological Society of London, Mr. Stanley reported a case which seems to come under this class. There were growths in the bladder, and an abscess in the middle line of the abdomen above the pubes. The abscess was opened just below the umbilicus, which was not patulous, and urine escaped mixed with pus. The patient, a boy aged two, sank from exhaustion a few weeks later ; in the interval between the operation and the patient's death no urine was passed naturally, all escaping from the wound. At the post-mortem, the opening in the abdominal walls was found to lead into an abscess between the recti and the peritoneum. At the lowest point of this abscess the end of the urachus was seen projecting from the fundus of the bladder. The tube of the urachus was free to the bladder, but no passage could be clearly traced from the urachus through the wall of the bladder into its cavity. I am here quoting from the original report written for Mr. Stanley by Dr. W. S. Kirkes, and preserved in the MS. "Report of cases in St. Bartholomew's Hospital," vol. v, p. 485. Dr. Morley Fletcher, who kindly found out for me the above report, after diligent search, has also turned my attention to specimen No. 2419 in the museum of St. Bartholomew's Hospital, where there is a sarcoma of the bladder, and an abscess cavity above it, extending to the umbilicus, "but no communication can be traced between the two (bladder

and urachus), although the urine continued to escape by the abscess up to the time of the child's death. The small papilla close to the vesical termination of the abscess is all that appears of the urachus." This specimen is most probably the same as Mr. Stanley's above described; it was reported on independently by Sir William Savory, and exhibited before the Abernethian Society in 1851, five months after Mr. Stanley showed it at the Pathological Society. Stanley in his MS. report believed that the sarcoma in the bladder caused obstruction, one consequence being that the urachus reopened. He further considered that the canal of the urachus ruptured, and the extravasated urine set up an abscess; but this theory is doubtful. More probably Wutz's valve was forced when the bladder was full of urine, as "the tube of the urachus was free to the bladder," but it could not be detected after death; indeed, its existence was not known in 1851, and it is not always distinguished with ease.<sup>1</sup>

I need not dwell on the commoner form of urachal fistula which opens at the umbilicus. Much has been written on this condition (Paget of Leicester, Jordan Lloyd, &c.). It must not be confounded with a diverticulum from other parts of the bladder. Boyer believes that in obstruction to the passage of urine through the urethra, a hernial protrusion of the mucous membrane of the bladder may develop and push up to the umbilicus. This theory seems doubtful when we bear in mind Luschka and Wutz's researches, which show the frequency of an open urachal canal. Mr. Henry Morris has published some instructive information on urachal fistula.

<sup>1</sup> Since this memoir was prepared, Mr. John Morgan has referred to the specimen, No. 2419, in his Lettsomian Lecture ('Lancet,' 1898, vol. i, p. 710).

*Cystic Dilatations of the Urachus.*

Much more pertinent to the present subject are the minute dilatations of the urachal canal noted by several observers. The indefatigable Wutz, whose care in investigating the foundations of the subject makes his opinions on more important questions presently to be discussed all the more reliable, succeeded in detecting exactly two dozen examples of cystic dilatation of the canal in his seventy-four post-mortem examinations to which I have already referred. They all lay in the lower third of the urachus, and varied in size from microscopic dimensions to the bulk of a bean (Fig. 5). They were lined by pavement epithelium, as a

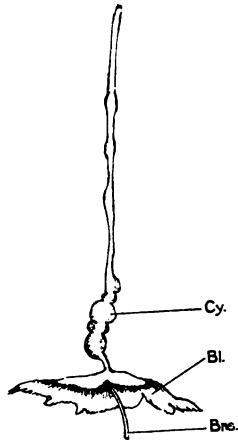


FIG. 5.—Cystic dilatations on the urachus, from a woman aged 66, who died of strangulated hernia (after Wutz). The canal here represented measured 3 inches. Immediately above the bladder (Bl.) a row of five small cystic dilatations (Cy.) bulge from the urachal canal. The largest was as big as a pea, their cavities communicated, and a fine bristle (Brs.) could be passed from the bladder through the cavity of the cysts, and for some way up the canal above them; the vesical orifice is guarded by a mucous fold.

rule stratified, and there was a capsule of plain muscle cells on the outer surface, a very important fact in relation to the microscopical examination of sections of the wall of a cyst suspected to be urachal (see Pl. VIII, fig. 1). Wutz

found inflammation and suppuration of these minute cysts in a case of fatal bladder disease in an old man and in a case of fatal peritonitis from perforation of the vermiform appendix in a youth aged twenty. This fact, that an abscess may develop in a small urachal cyst, must be borne well in mind. Supposing that it grows larger as an abscess, or supposing that a large urachal cyst suppurates, the epithelium would be destroyed, and probably the muscular capsule so altered as to escape detection. Hence an abscess in the middle line of the abdominal wall below the navel may well arise from inside the urachal canal, and the absence of the essential histological elements of urachal tissue does not always prove that a cyst in this region cannot have developed from the urachus. I may further suggest that a urachal abscess might burst and open up the subserous connective tissue. If it remained quiescent for some time, a clear fluid replacing the pus, it might readily be taken for a cyst of the urachus, and indeed would be a cyst of urachal origin; but no characteristic epithelium or muscular

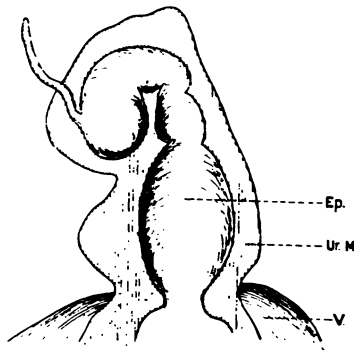


FIG. 6.—Cyst of urachus (Wutz, after Walter). Ep. Epithelial and subepithelial tissue of urachus irregularly dilated. Ur. M. Muscular coat of urachus dissected backwards. V. Bladder.

fibres would be found in its walls. The largest cystic dilatation in which Wutz believes is that described by Walter (Fig. 6).

*Cystic Tumours of the Urachus.*

As it is evident from the above researches that cystic dilatations of the urachus are by no means rare, it is reasonable to believe that they may develop into tumours capable of giving rise to marked clinical symptoms. Hence certain cystic tumours discovered between the abdominal parietes and the parietal peritoneum in the middle line below the umbilicus have been reckoned as urachal on very fair grounds. Yet, for various reasons, perfect evidence seems unobtainable in any one case. Wutz's researches were undertaken under every advantage for precise histological research; in operative practice these advantages are absent.

I will dwell a little on some of the most probable cases of "urachal cyst."

An instructive case is reported by Mr. George Morgan, of Brighton. A boy aged five years was admitted into the Children's Hospital with vomiting and diarrhoea, indicating, it was believed, peritonitis secondary to severe enteritis. Abdominal section was performed, and a cyst was found communicating with the bladder, so that urine escaped when its wall was opened. After careful dissection around the cyst the peritoneal cavity was exposed and flushed. The patient was too ill to allow of close examination of the parts, nor did Mr. Morgan attempt to dissect away the cyst wall, "but it was certain that the cyst was in the middle line running up to the umbilicus and communicating with the bladder." The cyst and bladder were washed out with boric solution; the wound in the cyst was closed with a double row of silk sutures, the stitches not penetrating to the mucous membrane. The abdominal wound suppurated, and pus and urine escaped for some days during convalescence, but the patient was well at the end of a month.

I place this case first, not only because it is relatively new (1896), but also because it seems transitional between

urachal fistula and urachal cyst. A typical fistula opens at the umbilicus and bladder. In Mr. Stanley's case the urachus or its suppurating cystic dilatation communicated with the bladder only. In Mr. Morgan's case we see a step further, for a definite cyst opened into the bladder. Let it be noted that here, as seems frequent in cases of patulous urachus, suppuration took place.

We now come to the typical cyst, which is cut off both from the umbilicus and the bladder. As its cavity is kept free from septic influences, suppuration seems rare.

Lawson Tait and Teichelmann's case might well be an instance of a genuine cystic tumour of the urachus. The patient was a married woman aged twenty-eight, who had miscarried twice, several years before the illness which brought her under observation. An attack of fever and vomiting occurred, a hard painful swelling developing between the umbilicus and pubes, somewhat to the left; at first there was dysuria. The general disturbance abated. The tumour filled the pelvis, and extended above the pubes in the middle line and to the left.

At the operation, three months after the attack of feverishness, the "tissues in the middle line were found thickened and cartilaginous in density." The tumour was a large cyst dipping into the pelvic cavity, which, indeed, it seemed to fill, but not adherent to its contents. "It was attached," the authors state, "by a short sessile (*sic*) pedicle, of more than two fingers' diameter, to the abdominal wall, somewhat to the left of the middle line and about two fingers' width above the pubes." The cyst was easily enucleated, the cavity drained, and the margins stitched to the abdominal wall. The peritoneal cavity was drained separately. The patient was discharged from hospital cured at the end of three weeks. This cyst seems to have been really outside the peritoneum, and from its position in the abdominal wall was most likely of urachal origin. Microscopical evidence seems wanting.

The next case is reported by Dr. Edward Ill, of Newark, New Jersey, in a valuable monograph on ' Tumours of the Abdominal Wall ' which appeared six years ago. I will reproduce the author's own words. " The patient had noticed a tumour of small size for about ten years, when it suddenly took on rapid growth during a pregnancy, and contained five litres of fluid by the third month. Incision and drainage cured her. She miscarried two weeks after the operation. This miscarrying after the operation is by no means rare (Tait and Roser). This very slow growth was noticed in one of Hoffmann's cases."

Here it is probable that the tumour arose from the urachus. Unfortunately Dr. Ill makes no note of the appearance of the wall of the cystic cavity when it was divided at the operation, nor does he say that he made out the peritoneal relations at the upper limit of the cyst wall, as I think could be done in my case ; still less could he approach so near to certainty as Dr. Douglas of Nashville, who in his case succeeded in dissecting away the cyst wall from the parietal peritoneum which lay behind it. Nor does Dr. Ill seem to have read Wutz's criticisms on Hoffmann's and Roser's so-called cysts of the urachus, presently to be considered, and he further appears to take for granted what Mr. Lawson Tait himself, as will be seen, admits to be theoretical.

A case of considerable interest was reported last October by Dr. R. Douglas, of Nashville, Tennessee. The patient was a sterile woman aged thirty-six, married eleven years. " About eighteen months ago she observed a swelling in the lower part of the abdomen, rather prominent on the right side. The enlargement was soft and painless. It grew slowly and did not materially show until the last four months, within which time its growth has been rapid, chiefly to the right side." The abdomen was asymmetrically distended to about the size reached at the seventh month of pregnancy, and chiefly to the right. It was soft, fluctuating, compressible, and fixed. The uterus was small, retroflexed, and low in the pelvis ; the

fluctuating swelling encroached on the anterior vaginal fornix. Diagnosis was uncertain.

At the operation, on cutting through the linea alba and transversalis fascia, Dr. Douglas exposed a "red, congested cyst wall," which he tapped, drawing off twenty-five pints of clear pale green fluid containing a few flocculi. As the sac collapsed it became evident that the peritoneal cavity had not been opened. The operator now felt sure that he had to do with a cyst of the urachus. It was easily enucleated, with but little hæmorrhage; the viscera could be felt through the parietal peritoneum exposed when the cyst was removed. The cyst wall dipped into the true pelvis in front of the uterus, which it thus displaced. It had no special attachment even to the bladder. A wide area of parietal peritoneum detached from the back of the cyst was simply left loose, the operator hoping that intra-abdominal pressure would bring it in apposition with the wall. Unfortunately it hung loose, and seems to have been the cause of the patient's death within forty-eight hours. The fluid was inadvertently thrown away. The cyst wall was thin and made up of "fibrous material showing no evidence of muscular structure."

This case seems as nearly genuine as the evidence available under the circumstances can make it. The cyst lay exactly in the site of the urachus, and was certainly not a diverticulum of the bladder. That it was extra-peritoneal there could be no doubt, for the serous membrane detached from the back of the tumour was found after death to be continuous with the rest of the peritoneum. Although no definite statement is made about the post-mortem appearances of the pelvic viscera, the author evidently implies that they were separate from the cyst, which could hardly, then, have arisen from the ovary or parovarium, burrowing forwards; microscopic evidence was negative rather than contradictory. No plain muscle-cells were detected, but the cyst wall had been greatly stretched for some time and apparently inflamed as well,



so that muscular tissue might have once existed, and undergone atrophy.

Granting that there can be cystic tumours of the urachus, it is not difficult to understand that they may incline very much more to one side of the middle line than to the other, for even the minute dilatations of the urachus already described are sometimes asymmetrical from the first.

*Evidence in the Author's Case.*

I will now explain how far the evidence was in favour of the theory that in my own case the tumour was a true cyst of the urachus.

The upper cyst, or loculus, occupied the greater part of the site of the urachus (Fig. 7). As far as could

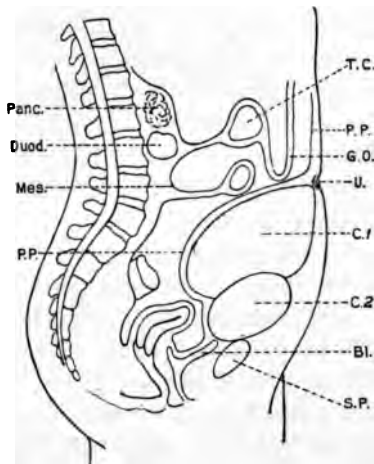


FIG. 7.—Diagram illustrating relation of the urachal cyst to the peritoneum (author's case). U. Umbilicus. C.1. Main cyst. C.2. Smaller cyst or loculus. Bl. Bladder, empty; a sound could raise fundus till it lay behind C.2. P.P. Parietal peritoneum pushed backwards by the cyst below the umbilicus. T.C., G.O., Mes. Transverse colon, great omentum, and mesentery, with small intestine displaced upwards by the larger cyst. Panc., Duod. Pancreas and duodenum. S.P. Symphysis pubis.

safely be ascertained at an operation, the appearances at its uppermost limits, already described, suggested that a true cyst wall and not an inflammatory peritoneal adhesion cut off the viscera and the peritoneal cavity from the parietes below the umbilicus. The posterior part of the cavity seemed to be lined by a definite regular wall, continuous with that in front, and could hardly have been mere organised peritoneal effusion concealing intestine.

The existence, position, and appearance of the lower cyst, or *loculus*, greatly added to the probability that the whole tumour was urachal. It had a very definite wall bulging prominently upwards from the connective tissue in front of the bladder into the cavity of the larger cyst. When I exposed it I thought that it might be a small encysted dropsy enclosed in the fold made by the parietal peritoneum passing on to the bladder, but it is difficult for fluid to collect in that fold, and I have never seen nor ever heard of an authentic case of a cyst thus developed. An encysted dropsy so perfectly bilocular is highly improbable. The prominent well-developed septum, or upper part of the lower cyst, was very unlike an inflammatory product, but closely resembled the septa seen in entirely neoplastic cystomata and in cysts due to congenital malformations. In short, it seems reasonable to believe that the two cysts were developed from the bead-like dilatations detected by Luschka and Wutz (Fig. 5) in the urachus in many post-mortem examinations.

The strongest evidence, however, that the cyst arose from the urachus was afforded by microscopical examination of sections of the cyst wall. Mr. Shattock found that the wall was largely made up of plain muscular fibre (Pl. VIII, fig. 1); and Luschka (Pl. VIII, fig. 2) and Wutz have both proved that there is a muscular coat to the urachus, very conspicuous in the sections of normal urachus exhibited this evening (Pl. IX, figs. 3 and 4). Wutz further discovered a special muscular capsule to the minute cystic dilatations already described.

The anatomical relations were made at least sufficiently clear at the operation to prove that the cyst wall could not have been derived from the broad ligament.

The loose flocculi which floated about in the cyst were carefully examined under the microscope by Mr. Keep, who could not find any hooklets or other elements detected in hydatid cysts. The presence of the well-defined loculus, or second cyst, showed that the tumour could hardly have been an old abscess cavity due to anterior parametritis or to direct injury of the parietes in the middle line.

Other cases similar to those just described have been related, but I need not multiply examples. Dr. D. Newman's case ("Cyst of Urachus," *Glasgow Med. Journal*, vol. xliv, 1896, p. 20) was like G. Morgan's, the bladder opening into a urachus not pervious at the umbilicus, so that it became greatly dilated. When the patient was thirty-nine he underwent operation. The cyst, or dilated urachus, was drained, urine escaping for about a month, then it closed completely. Rotter (*Urachus-cyste mit Carcinom der Blase*," *Deutsche med. Wochenschrift*, Jan. 20th, 1898, Supplement, p. 10) dissected out a large urachal cyst, with the parietal peritoneum adhering posteriorly, and then found that its walls below were continued into the vesical walls. At the point of junction was a small villous cancer of the vesical mucous membrane already diagnosed by the cystoscope. He excised the cancer with the contiguous part of the bladder and the whole urachal cyst. The wound in the bladder and the abdominal incision were carefully closed, and the patient recovered speedily. The relations of the cyst, so evident in this case, seem to leave no doubt as to its origin.<sup>1</sup>

Sutton and Aveling's extra-peritoneal tumour, described in the second volume of the *British Gynaecological Journal* (p. 187), was a myo-sarcoma of the muscular sheath, not a cyst developed from the urachal canal.

<sup>1</sup> See Addendum, p. 385.

*Tait's so-called Allantoic Cysts.*

Over ten years since, Mr. Lawson Tait published a report of high importance on a series of cases where a capacious cystic cavity was found occupying the middle and lower part of the abdomen and the whole of the pelvis. He maintained that it was extra-peritoneal, and represented an extreme form of urachal cyst. He ventured further on a very bold and ingenious theory, though it must not be forgotten that he has never professed that it was more than a theory. "I think I have a satisfactory explanation to give of them" is the most definite statement he makes concerning his cysts. Dr. Byron Robinson, the chief upholder of the theory, is almost as cautious. I cannot find any corroborating evidence since the original reports of Mr. Tait's cases were published by the operator and by Dr. Robinson.

The theory is not very difficult to understand, and as Dr. Robinson endeavoured to make it clear by a diagram, I have prepared a sketch which perhaps illustrates the relations of cyst and peritoneum yet more completely (Fig. 8). Mr. Lawson Tait believes that the first change is arrested development early in embryonic life, when the allantois is growing out of the peritoneal cavity. The allantois ceases to be a part of that cavity, and does not contract into the vesicle into which it normally dwindles. Here it is noted that the umbilicus always seems to open straight into these large abdomino-pelvic cysts. The peritoneum can develop normally in its upper part, but it is arrested inferiorly by the persistent allantois. Hence according to this doctrine the upper viscera receive their normal serous investment by pushing forwards the peritoneum in front of them.

The pelvic viscera, however, which lie entirely below the peritoneum push forwards the posterior and under-part of the allantois and thus make for themselves serous folds out of the allantois itself. As Mr. Tait says, the

cyst which goes down into the pelvis is, according to his theory, developed from the allantois, and acts as peritoneum for the pelvic viscera. No other peritoneum, he insists, ever entered the pelvis in his cases.

Twelve cases of these large cysts have been described by Mr. Tait, and Dr. Robinson adds some more observed in

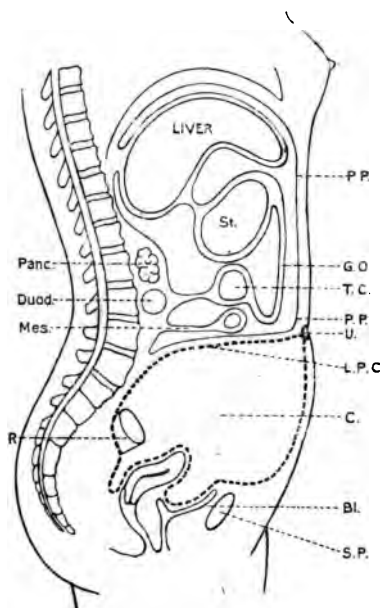


FIG. 8.—Diagram illustrating Lawson Tait's and Byron Robinson's theory on relations of large "allantoic cysts" to the peritoneum. C. The "allantoic cyst" dipping into the pelvis. Its wall is indicated by a dotted line, and it is seen reflected, according to the theory, over the pelvic viscera, after the manner of normal peritoneum. U. Umbilicus. P.P. Parietal peritoneum. L.P.C. Limits of the peritoneum and cyst wall, the former not extending, according to the theory, below the level of the umbilicus. T.C., G.O., Mes. Parts displaced as in Fig. 7. Bl. Bladder, R. Rectum. S.P. Symphysis pubis.

Mr. Tait's practice. The cases themselves have been repeatedly quoted, and cannot be discussed here at any length. But of the original twelve recorded by Mr. Tait himself, three died from the operation, and a fourth died

just a month afterwards from miscarriage, being pregnant when operated upon. Unfortunately there was no necropsy on any of the four. These cases were no doubt very ill, and the justifiability of an exploratory incision need not be disputed. At the same time all seem to show evidence of much more pathological mischief than could be accounted for by a purely teratological condition, which does not imply inflamed tissues or acute disease and which let the patients live in comfort to adult life.<sup>1</sup>

The same can be said of the eight other cases which recovered. The common features were the existence of a spacious cyst-like cavity in the abdomen and pelvis, and the alleged absence of peritoneum between the transversalis fascia and the cyst wall. The operator noted in several cases how the peritoneum left the abdominal wall about or above the level of the umbilicus, passing back over the top of the cyst, whilst the cyst wall lined the pelvic viscera and bladder as peritoneum (Fig. 8 explains this alleged condition). He declares how, in one case, the cyst wall seemed reflected over the ovaries and tubes, whilst the intestines lay in a pouch of peritoneum on the top of the back part of the cyst immediately above the level of the sacral promontory,<sup>2</sup> just as they are normally seen much lower down in Douglas's pouch. The writer signifies, no doubt, the small intestines, for if his theory be true the sigmoid flexure and part of the rectum would, after the fashion of the uterus and tubes, push forwards for themselves an allantoic serous investment, so that there would be an allantoic Douglas's pouch in the normal position of that fold, far below the pouch formed by the lowermost limits of the curtailed true peri-

<sup>1</sup> I find that Mr. Henry Morris (*loc. cit.*, p. 1067) observes, "It is a remarkable thing, if the theory of the urachal origin of these cysts is correct, that the cysts remained so many years in abeyance, and then suddenly developed to such an enormous size."

<sup>2</sup> This pouch would be the posterior inferior extremity of the peritoneal sac indicated under the mesentery (Fig. 8). If full of intestine it would press against the promontory of the sacrum.

toneum. Whilst this latter, or false Douglas's pouch, is described as above related in one case, the ninth, Mr. Tait states that in the tenth the cyst wall was reflected over the pelvic organs, the intestines behind the posterior cyst wall being attached to it by connective tissue, as where there is normally no peritoneum. It is implied that this connective tissue is not an inflammatory product, not old adhesion in fact. The observation is very interesting, but if correct would represent a condition not explained by the allantoic theory. Where was the lower limit of the true peritoneum in this case? How came the intestines to be devoid of peritoneum? or if they be allowed their mesentery and serous coat, how came they to lie outside both the parietal peritoneum above and the allantoic cyst wall below?

Most of these cysts contained great quantities of fluid, one as much as thirty pints. Broken-down lymph and amongst other things masses of fat like omentum were found in the fluid. Byron Robinson examined similar cases in Mr. Tait's practice and found flaky flocculent lymph shreds and gelatinous masses, or even thick pus, in the cavities of these great cysts. As for the cyst wall, it was often as "thick as sole leather," very easily torn, and gelatinous, unlike peritoneum, whether it lay against the transversalis fascia replacing (as was believed) the parietal peritoneum, or lined the pelvic viscera in the manner already described. These appearances will be further considered.

Undoubtedly the large cysts described by Mr. Lawson Tait represent a remarkable condition, very puzzling to the operator. Mr. Tait deserves great credit for describing them, and Dr. Byron Robinson must be thanked for giving a clear demonstration of the "allantoic cyst" theory. But the evidence that these cysts are urachal remains defective. I will now consider some reasonable objections.

1. Tait and Robinson definitely state that the allantoic origin of these cysts has not been verified by post-mortem

dissection in any of their cases. Bland Sutton does not distinctly accept the theory.

Mr. Tait's words are, "It remains quite uncertain what the exact nature of the tumours really was, though I think I have a satisfactory explanation to give of them."

Dr. Byron Robinson declares that "it must be admitted that we have so far no conclusive scientific post-mortem of cases in which the urachal cyst completely fills the whole pelvis, and until such autopsy is produced with frozen section, we must wait for light."

Mr. Bland Sutton writes, "Lawson Tait has published details of allantoid (urachus) cysts that have obtained large dimensions. . . . At present so little is known about the cysts that it is impossible to decide as to the best method of dealing with them." This distinguished pathologist has made some instructive observations on urachal cysts and their comparative anatomy; but the words just quoted were written several years after his own report of microscopic appearances seen in Mr. Tait's cases; this report will presently be discussed.

2. Wutz, who wrote earlier than the above-named authorities, demonstrates grave errors in the post-mortem reports of cases with similar symptoms described as "cysts of the urachus," and in the clinical records of other cases, so described, which recovered.

Thus out of Hoffmann's three cases of supposed "cysts of the urachus" repeatedly quoted, in one the urachus was simply pervious throughout in a man aged twenty-eight, and in the second the cyst lay in the upper part of the abdomen and was most probably hepatic. There remains the famous case often referred to in the allusion that "cysts of the urachus have been known to contain fifty litres of fluid." Wutz fifteen years ago wrote a very depreciatory report of this case.<sup>1</sup> The patient was a man aged

<sup>1</sup> Freer (as well as others) has clearly overlooked this report. He relates Hoffmann's case (*loc. cit.*, p. 111), correctly noting that the fifty litres were found in the cyst after death, but accepts Hoffmann's interpretation unreservedly.



twenty-six ; the symptoms reasonably led to the diagnosis of chronic peritonitis. He died after repeated tapplings, and the famous fifty litres were discovered not at an operation but at the necropsy. The value of the original post-mortem report as an accurate guide is indicated by Wutz when he comes to a passage in that document which states that the wall of the cavity was reflected "over the excavatio recto uterina." Here he adds his own note in parentheses " (Man !)." He further, having examined the specimen in a museum, finds no evidence that the cyst wall was extra-peritoneal at any point. It may here be noted that the wall was originally reported as lining the under surface of the liver ; in Pratt's case, to which I must again refer, the same condition was detected. Above all, Wutz found that the urachus was quite separate from the cyst, and only contiguous to the outer part of its wall near the umbilicus. He considers, in fact, that Hoffmann's famous "urachus cyst" was simply a case of chronic hæmorrhagic peritonitis. Let writers beware of second-hand information, especially when in the original there is talk about the recto-uterine fold in a male subject.

Wutz's comment on Heinecke's case is especially important, as according to the original it resembled a "Tait's cyst." On tapping "pavement epithelium in a state of fatty degeneration" was obtained, and on the strength of that discovery "cyst of the urachus" was diagnosed. Hence the operator was prejudiced. On abdominal section no peritoneum was found, and the posterior wall of the cyst was clearly a very thin membrane covering coils of gut. At the necropsy the wall of the cyst was found lining the pelvic viscera, just as in Tait's cases ; it likewise lined the under surface of the liver, as in Pratt's and Hoffmann's cases. The cyst was seen to be an encapsuled peritoneal exudation.

Wutz criticises the report of Roser's case, repeatedly quoted as an instance of urachal cyst. A cyst communicated laterally with the bladder ; it was incised, and after

a time a urinary fistula developed. Wutz remarks that the cyst might have been ovarian, or an old peritoneal abscess communicating with the bladder or a vesical diverticulum. The patient was apparently lost sight of, and nothing could be verified.

Lastly, Wolff's case is also very uncertain. A tense cyst lay on the left side of the hypogastrium in a woman aged thirty-one. It was found at the operation entirely in front of the peritoneum, and was enucleated whole, but its base apparently lay deep in the left iliac fossa. There was no pedicle. Plain muscle cells were found in its outer wall. From the situation of its deeper part Wutz strongly suspects that it was a parovarian cyst which had burrowed in front of the peritoneum. The case recalls another related by Mr. Knowsley Thornton. He operated in 1878 for removal of a fluctuating, somewhat flat swelling, which chiefly occupied the left side of the abdomen; the patient was a woman of thirty-two, almost the same age as in Wolff's case. He came upon what he believed to be a cyst with universal adhesion over its anterior surface, and could not feel certain whether he had cut through the parietal peritoneum or not. After exposing and tapping a cyst full of clear watery fluid, he was able to shell it out from its capsule entire. A cavity was left which passed to the bottom of the pelvis. The uterus could only be felt through a thick membrane, and no other viscera were exposed, but there was yet another large cyst. This was tapped and enucleated; the cavity whence it had been shelled out was separated from that which the first cyst had occupied by a firm membrane. The intestines could be felt through the walls of this cavity. The operator scratched through some tissue at the bottom of the pelvis and reached the left ovary, full of blood and lying in a space cut off by adhesions from the general peritoneal cavity. The patient recovered. The resemblance to Wolff's case is evident; it is equally clear that an operator prejudiced in favour of urachal cyst might have assigned

an allantoic origin to the two tumours. They clearly are not examples of Tait's large cysts, whatever the latter may pathologically be, for they were readily enucleated.<sup>1</sup>

3. The evidence of anatomical relations so far as they can be gleaned at an abdominal section is clearly insufficient to settle the allantoic nature of a "cyst" of this class. Thus, amongst other obscure points, it is impossible to prove at an operation that the peritoneum (diseased or healthy) is replaced in the pelvis and lower part of the abdomen by the wall of a supposed "allantoic cyst." It is likewise impossible to prove at an operation that the intestine is attached to the upper and outer limits of the cyst, neither by inflammatory adhesions nor by the inferior reflection of an abnormally short peritoneum, but by connective tissue, presumably subperitoneal as in Mr. Tait's tenth case noted above. Only a very careful dissection or a successful frozen section could settle so important a matter. The question of the reflection of the "allantois" over the lower viscera will be discussed presently.

4. The development of a urinary fistula after drainage of an "allantoic cyst" (Tait, Case 6) seems at first sight to prove that the cyst must be connected developmentally with the bladder; but I have known of urinary fistulæ developing after or during abdominal drainage in cases of chronic, especially tubercular, peritonitis.

In Tait's second case, which was pregnant, a large cyst was found, adherent everywhere. Seven pints of putrid urine were evacuated. As at my operation, an instrument was passed into the bladder, but no communication with the cyst was detected. A urinary

<sup>1</sup> As it is practically certain that they were not urachal cysts, I need not enter into Mr. Knowsley Thornton's theory that they developed in the peritoneal subendothelial tissue. I have published my own experience in "Cap-sules, Real and False, in Ovariectomy," 'Brit. Med. Journ.,' 1896, vol. i, p. 960. No. 4, fig. 3, in that communication illustrates a "cyst of ovarian origin burrowing in the lower part of the broad ligament, leaving the mesosalpinx intact." Such a cyst may rise forwards and push up in front of the parietal peritoneum.

fistula developed three weeks after the operation, and the patient died a week later from miscarriage. There had been violent pelvic pain, sudden abdominal swelling, and retention of urine for twenty-four hours, a mass developing on the left side two days later. Then the operation was performed. This case is very interesting, but does not require an allantoic cyst theory to explain it.

5. It is unscientific, I must admit, to bring forward embryological theories to support or attack some particular interpretation of pathological appearances, but after all we know something about the development of the allantois. The portion retained within the abdominal cavity becomes bladder and urachus. In these great cysts alleged to be allantoic, not only the urachal portion remains open, but that part of the allantois which naturally remains outside the foetal body becomes, according to Tait's theory, included within the abdomen. How comes it then that the bladder, also a part of the urachus, remains normal, according to clinical and surgical evidence? In cases of quite trifling dilatation of the urachus verified by dissection, the bladder not rarely communicates with the canal of the dilated foetal relic

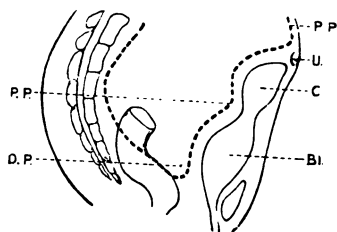


FIG. 9.—Urachal cyst communicating with bladder, in a boy aged 10, showing relations determined after death (after Byron Robinson). C. Urachal cyst. Bl. Bladder. U. Umbilicus. P.P. Parietal peritoneum pushed backwards by the cyst below umbilicus as in Fig. 7. D.P. Douglas's pouch.

(Fig. 9), and the same communication is frequent in fistula of the urachus opening at the umbilicus. If

Tait's cysts were allantoic, then, judging from what is seen in these less defective conditions, surely there would be ectopia vesicæ or even ectopia of the abdominal viscera. At the very least we must expect something like a vesical fistula. Ectopia of the viscera is essentially due, no doubt, to failure of closure of the visceral folds, but such extreme malformation of the allantois would gravely impede or even prevent that closure. This malformation, in order to meet Mr. Tait's theory, must be established early in foetal life, when it is conceivable<sup>1</sup> that the intestines and pelvic viscera could push forwards against an over-developed "allantoic cyst" and become invested by its wall, as they would have normally been invested by peritoneum. These changes, however, are fairly advanced before the abdominal walls have closed in anteriorly, and the abnormal allantois would be in their way. The well-known relations of the allantois to the placenta seem altogether ignored by Tait and Robinson. These relations must suggest to all of us manifest objections to the theory, which it is for those who advance it to explain.

Mr. Bland Sutton has put forward a more logical theory as far as the development of the normally internal portion of the allantois is concerned. "Instead of a portion of the allantois narrowing to form a urachus, the whole of its *intra-abdominal portion* may dilate and form a large *urinary bladder*." The words which I have emphasised in this quotation show that his theory is different from Tait's, and would represent a different type of cyst; for according to Tait the external part of the allantois would appear to become included in the abdomen, whilst according to the same observer's surgical experience the urinary bladder always managed to reach its full development, and was leaky in one case only (No. 2). Sutton quotes Mr. Shattock's case, where the abdomen of a four-months' foetus was filled by an

<sup>1</sup> Conceivable I admit, though we must not forget that it has never been proved.

enormously dilated bladder. In this instance, however, the urethra was imperforate. There is no question of any imperfection of the urethra in Mr. Tait's cases, nor does he base his theory on any such malformation. Urethral obstruction, it is well known, may open up a urachal fistula.

To return to Mr. Tait's views ; Reginald Pratt's case, where the cyst wall appears to have lined the under surface of the liver, is of high surgical interest, but would appear to be a *reductio ad absurdum* of the allantoic cyst theory. It can be met as such by objection 3, and especially by the argument which I have just advanced about ectopia of the viscera. But Wutz refers to cases, clinically similar, where dissection showed that the "cyst wall" was probably diseased peritoneum, and Dr. Cone of Baltimore's case, as will be shown, throws further light on possible fallacies attending the allantoic *pseudo*-peritoneum theory.

6. Microscopic evidence is unsatisfactory in Tait and Byron Robinson's cases, and is almost bound to be negative (in the sense expressed above in my remarks on the simple type of cyst) in such cases where disease was so advanced, and avowedly complicated in some instances by tubercle. But the evidence was not merely negative, it was sometimes contradictory. In Tait's fourth, seventh, and twelfth cases fragments like omentum were detected inside the cyst. In the twelfth, indeed, Bland Sutton reported one fragment as "omentum," and in the others they very possibly were the same, or else were "masses of congealed fat" such as Cone of Baltimore found in the peritoneal cavity in a case of encysted dropsy of the peritoneum secondary to tubercular disease of the tube and ovary. If so, in either case the "cyst" was simply an encysted peritoneal effusion. In Tait's first case it is noted that "mucoid epithelium" was found on the inner wall. No drawings are given, nor is it stated what is meant by "mucoid" epithelium. Mr. Bland Sutton's evidence in Tait's eleventh case is also inconclusive.

"By scraping, Mr. Bland Sutton obtained numbers of flattened rounded and pyriform cells, similar to those found in the mucosa of the urinary bladder, though very much smaller. But the patient in this case was a girl of seventeen, with high pulse and temperature. Mr. Tait before the operation suspected tubercular peritonitis, and I cannot help thinking that his diagnosis was correct, and needed not to be replaced by an allantoic cyst theory. The cells obtained by scraping might have been tubercular, identical with the "lymphoid, epithelioid, and polymorphonuclear" cells detected in sections of the serous coat of the uterus in Cone's case of tubercular peritonitis. This recalls to our memory Byron Robinson's researches on a tube and ovary which Lawson Tait had removed from the interior of a so-called allantoic cyst. Dr. Robinson believes, consistently with Tait's theory, that the allantois was reflected over the tube, forming an allantoic instead of a peritoneal broad ligament. This opinion, however, becomes questionable when we compare his description with Cone's account of the serous coat of the uterus in a tubercular case, and find that Robinson admits that "some tubercles with giant cells were visible." Whilst believing that the cyst wall, though of allantoic origin, served as peritoneum to the pelvic viscera, Robinson observes, "However, I must say that this cyst wall does not look much like peritoneum to me, even after making due allowance for pathological changes. In the cases I saw it was as thick as sole leather, friable, brittle, gelatinous, and reflected light when it was torn or cut, just as jelly would." Then he speaks of finding tubercles and giant cells in the mesosalpinx, which he believes to be in these cases an allantoic, not a peritoneal fold.

All this evidence, however, would seem to indicate not only that tubercular disease was present, as is more or less admitted, but further that the "cyst wall" was simply true peritoneum greatly altered by tubercular disease, and covered more or less uniformly with a thick layer of tubercular deposit.

In Cone's case of tubercular peritonitis, so carefully explored after death, this change produced "an opaque, yellowish-white, felt-like membrane" about a tenth of an inch thick, which was reflected over the pelvic viscera, "forming a complete blanket." In another case in my own practice I found a thick greenish membrane lining Douglas's pouch, and following the reflections of the pelvic peritoneum. This membrane was simply tuberculous matter incorporated with the peritoneum; there had been tubercular disease before, and the patient suffered afterwards from tubercle elsewhere. In short, the affection which I discovered was tubercular peritonitis in a half quiescent condition; fluid full of cheesy matter had collected, and simulated a cystic tumour of uncertain origin. In another chronic tubercular case I found the parietal peritoneum converted into a thick, yellow, spongy membrane. Thus it is quite reasonable to suppose that Dr. Robinson's cyst wall "as thick as sole leather" was identical with the membranes which Cone and myself observed, as many others must have observed, in cases of undoubted tubercular peritonitis; in fact, it was tubercular peritoneum. The character of the cyst wall in these cases not only suggests that disease, but also compels us to put aside the supposition that the cyst represents an old, quiescent, serous perimetritis, such as Matthews Duncan and Sir John Williams have described. Hence the latter affection need not be discussed, although it is quite conceivable that a serous perimetritic cyst might puzzle an operator, and induce him to ascribe to it an allantoic origin.

7. From the foregoing objections we may conclude that these so-called "allantoic cysts" were really encysted dropsies due to chronic peritonitis, usually if not always tubercular. For encysted dropsy is often of tubercular origin, and liable to continue long after the subsidence of active tubercular disease, as the peritoneum is much damaged and hence has lost its power of rapid absorption. The fluid naturally tends to collect in



Douglas's pouch, if that cavity be not effaced by adhesions, to rise forwards and to push the small intestines upwards. The parietal and visceral peritoneum bounding the effusion is very slow to return to its normal appearance. Recurrence of acute symptoms is always possible. When it occurs it is natural that the surgeon should explore, and when he has opened the abdomen the appearances are very puzzling. The peritoneum is completely altered, not only from its normal state but also from its familiar appearance when studded with recent tubercular deposit. As there was a great collection of fluid in Tait and Robinson's cases, and as tubercle, on their own admission, complicated more than one, I cannot help thinking that most if not all might be examples of old encysted dropsy due to tubercular disease. This explanation seems simpler than their own, which they admit to be theoretical, and Wutz's scepticism as to the urachal origin of large cysts must still be respected as eminently rational and never as yet refuted by clinical research or by surgical experience.

#### *The Surgery of Urachal Cysts.*

Experience teaches us that what are termed "cystic tumours of the urachus" in this communication may often be dissected away with comparative ease (Tait, Douglas), whilst in other cases (Ill, Author) extirpation is impossible and incision and drainage answer well.

When a cyst has been dissected away from a large area of peritoneum it is best to trim the detached serous membrane, even if it appear unhurt. In Douglas's case it seems clear that sloughing of the flap was the cause of death. The peritoneum should be cut away until it is even with the cut edges of the parietes in the abdominal wound; then sutures can be applied as in an ordinary abdominal section. If, however, the peritoneum has been too extensively damaged to allow it to be neatly adjusted to the wound, it should be freely trimmed away

up to the line where it joins the parietes. There is no danger in leaving a large area of the parietes, free from peritoneum, facing the peritoneal cavity. I have often had to face this condition after the removal of strongly adherent ovarian and other tumours, and never seen evil results. The parietal peritoneum is relatively more damaged in the case of a urachal cyst, as it is uniformly torn from its normal attachment; in ordinary abdominal operations bruising of its free surface is the usual injury, and more likely to be followed by oozing than sloughing.

The operator should never run risks through an extreme anxiety to leave the peritoneal cavity unopened.

Especial care must always be taken to ascertain the relations of the bladder to any cyst which might, by its position, be urachal. A sound should always be passed into the bladder by an assistant when the lower part of the cyst is being handled. Should a communication with the cavity of the bladder be discovered (Fig. 9), it is almost needless to say that it must be closed. The sutures should not include the vesical mucous membrane. Similar treatment is demanded if the bladder be torn during the separation of the cyst wall. Bearing in mind Wutz's anatomical researches, the operator must never forget that a valvular communication between the urachal canal and the bladder seems to be the normal condition when the canal is pervious (Fig. 5), nor must he forget that the wall of the bladder may be damaged at the operation and not give way till later on. Hence he must be on the look-out for urinary fistula for long after the patient's apparent recovery from the operation. Ill states that he once saw a case where a communication between the bladder and the cyst was overlooked or imperfectly obliterated. "The odour from the patient's sore was indescribable, and made life a torment to himself as well as his surroundings."

The abdominal wound should always be free, so as to extend as far downwards as the bladder and so far upwards

as to pass well above the superior limits of the tumour. The surgeon can never proceed safely till he has made sure of the relations of the tumour. Their importance below is evident, above it is very necessary to examine the condition of the intestines and the peritoneum.

When the cyst wall cannot be detached it must be freely packed with gauze, or simply drained, as the case may be. The parietes must be closed from above downwards; the lower end of the wound being left open to allow of the removal of the tube or dressings. The tube must not be suffered to press on the bladder.

The enormous cyst-like cavity which Tait and Robinson explain by the allantoic theory is chiefly remarkable to the surgeon for the extreme perplexity which it causes him when he opens the abdomen for exploratory purposes. I have dwelt enough on this kind of cyst; whatever it may be, the authorities who have written on it dissuade us from any attempt to enucleate its wall. Personal experience makes me very disinclined to drain when I discover anything approaching tubercular peritonitis. That malady is best treated by free drying of the peritoneum and closure of the abdominal wound. Mr. Tait's "circular drainage," conducted through the abdominal wound and also through Douglas's pouch, might be reasonable enough if a true urachal cyst, not tubercular, managed to bulge downwards and backwards behind the uterus, adhering to the lowest part of the pelvic peritoneum.

#### ADDENDUM.

The Council of the Society have kindly permitted me to add two unpublished cases of cystic tumour of the urachus related by the President in the discussion which followed the reading of the above communication.

Mr. Bryant observed that, in the first case, ovarian dropsy was suspected. He continued: "In making my incision, however, through what I believed was the

abdominal wall, I suddenly came down upon a cyst, which I opened, thinking I was dividing the peritoneum. To my surprise I came into the cavity of a cyst which discharged a quantity of fluid, not of the thick mucoid character with which we are familiar in ovarian disease, but of a thin serous nature, more or less blood-stained. I did not, however, think much of the fluid at the time, although I thought I had opened the cyst too soon. I then drained the cyst, and in following it upwards by way of its removal, to my surprise I found on tracing its posterior wall that I had not yet opened the peritoneal cavity. I consequently enlarged my opening a little upwards and came upon the peritoneum, the cavity of which I opened. I put in sponges to keep the intestines, &c., well out of the way, and traced the peritoneum, which was reflected from above the umbilicus on to the posterior part of the cyst. I proceeded subsequently to take the cyst away, being all the time somewhat puzzled as to its relations. In dissecting it off I found it was closely connected with the bladder, so much so that I was alarmed lest I should wound that organ. I therefore passed a sound into the bladder as a guide, but it was with considerable difficulty that I could separate the cyst from the fundus of the bladder. I succeeded, however, in doing so, and I was then surprised to find that the cyst fell into my hands; in fact, there was no pedicle, so that the cyst was obviously not ovarian. If I had possessed the knowledge which the author has placed before us to-night I have no doubt I should have made a thorough diagnosis. I remember also that the wall of the cyst was exceedingly thick and fibrous. The case eventually did well."

As the cyst lay in front of the peritoneum, was enucleable, yet closely connected with the bladder, there can be little or no doubt that it had developed from the urachus. Surgically, this case resembled Douglas's, Rotter's, and Tait-Teichelmann's, where the cyst could be and was enucleated. In the close relation of the cyst to the

bladder this case recalls Rotter's and my own, as there was no difficulty in separating the cyst from the bladder in Tait's and Douglas's. The cyst in Mr. Bryant's case was not bilocular as in mine, nor complicated with cancer of the adjacent part of the bladder as in Rotter's.

Mr. Bryant's second case was undoubtedly an example of urachal cyst in a male. "In the early seventies, a man aged about thirty-five came under my care at Guy's Hospital complaining of great pain in the abdomen, with a swelling extending from the umbilicus to the pubes. He had a high temperature, and the tumour was extremely tender to the touch. To my mind it seemed certain that an abscess was forming behind the abdominal parietes. The bladder appeared to be all right. We drew off some urine which was quite natural, and we noticed at the time that the penis was not properly formed, there being slight hypospadias. On going into the history of the case the man told me that he had for years noticed a lump below the navel 'about the size of a small cocoa-nut.' It had never troubled him much, but was sometimes larger than at others. The patient, who was an intelligent man, thought it was the lump in question which was the seat of his trouble. By-and-by it became necessary to open it, and when I did so a most horribly fetid material escaped, which did not smell like urine. When the contents of the cyst were evacuated the cyst wall was found to be thick, and covered with lymph and sloughing tissue. After I had washed it out with iodine water and made it sweet, I still could not detect any smell suggestive of urine. I plugged the cyst with lint soaked in terebene oil, and put a few sutures into the large wound I had made. The plug was left in for some days, and on removing it no marked urinous smell could be detected. At the second or third dressing, however, the patient assured me that some urine had escaped from the wound, and close observation confirmed this statement. There was, however, no bladder complication; the wound subsequently contracted up, and in

a few weeks the patient made a good recovery. I, at the time, recorded that case as one of ulceration of the fundus of the bladder into the connective tissue, causing a perivesical abscess. I am sure now that it was one of urachal cyst."

This case is of the same type as George Morgan's and Dr. Newman's, representing an intermediate condition between urachal fistula and urachal cyst. As in Newman's the patient was a male adult, and as in his the communication with the bladder appears to have closed spontaneously. The forcing open of Wutz's valve through over-distension of the bladder may account for this transitional form of cyst. It would be well if some anatomist would further investigate the anatomy of this valvular fold at the vesical orifice of the urachus. We cannot feel sure that it is constant, yet if not constant it may none the less represent a normal condition, its absence being in that case abnormal. The surgeon dealing with a cyst of this kind must bear in mind Ill's experience of a secondary urinary fistula following an operation by another surgeon for urachal cyst.

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<sup>1</sup> In this, the earlier of the two works by Mr. Sutton named in this table of references, it is noted that Mr. Tait believes that his large cysts originated in the urachus, and the reader is referred to that surgeon's paper in the 'Brit. Gynec. Journ.,' vol. ii, "for further information." With scientific caution, Mr. Sutton concludes, "For my own part, I believe the cysts are commoner than many suppose, but are unrecognised; and, what is more important, there is very good reason to believe that certain rare cases of cystic tumours, of large size, occasionally found between the peritoneum and the sheath of the rectus muscle, take origin in this structure." Mr. Sutton gives a good summary of the opinions of German authorities on the comparative anatomy of urachal cysts.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 142.)





## DESCRIPTION OF PLATE VIII.

### A Case of Cyst of the Urachus; with Notes on Urachal and So-called "Allantoic Cysts" (ALBAN DORAN).

FIG. 1.—Microscopical section of wall of cyst of urachus. (Author's case,  $\times \frac{1}{2}$ , Beck.) Plain muscle cells in longitudinal view are seen below. Above similar cells appear in transverse section, and some divided obliquely appear unusually large.

FIG. 2.—The urachus (after Luschka) from a man aged 50. Outer aspect of vesical mucous membrane (*a*) is exposed; muscular coat (*b*) turned back. The muscular sheath (*c*) of the urachus, tendinous above (*d*), has been divided longitudinally and turned back. The urachal canal (*e*) is exposed, showing bulbous dilatations (*f*).



FIG. 1.

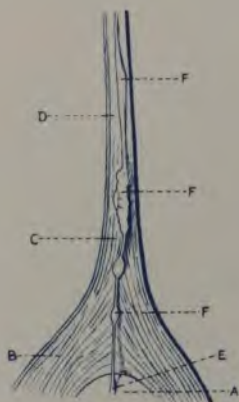


FIG. 2.





## DESCRIPTION OF PLATE IX.

### A Case of Cyst of the Urachus; with Notes on Urachal and So-called "Allantoic Cysts" (ALBAN DORAN).

FIG. 3.—Longitudinal section of the normal urachus midway between the umbilicus and bladder,  $\frac{1}{8}$ " obj., showing well-marked plain muscle cells seen at full length. (From a woman aged 45, subject to myoma of the uterus.)

FIG. 4.—Transverse section of the same (see fig. 3). Groups of plain muscle cells are seen divided transversely. White fibre, representing the tendinous part of the urachus, is abundant. The canal has entirely disappeared. ( $\frac{1}{8}$ " obj.)

PLATE IX.

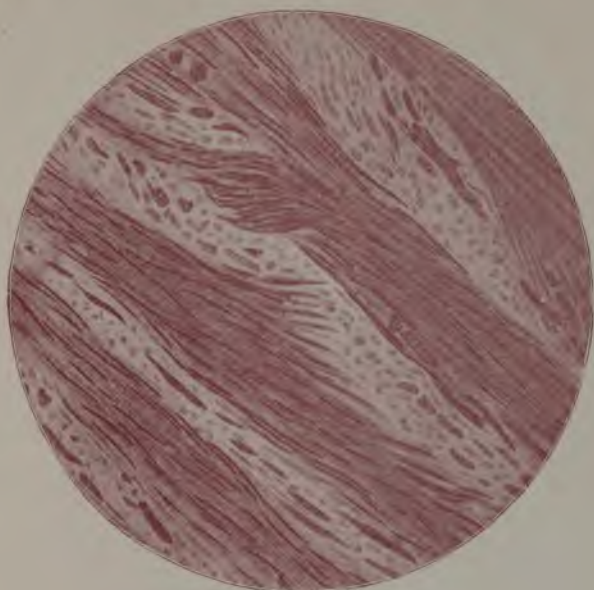


FIG. 3.

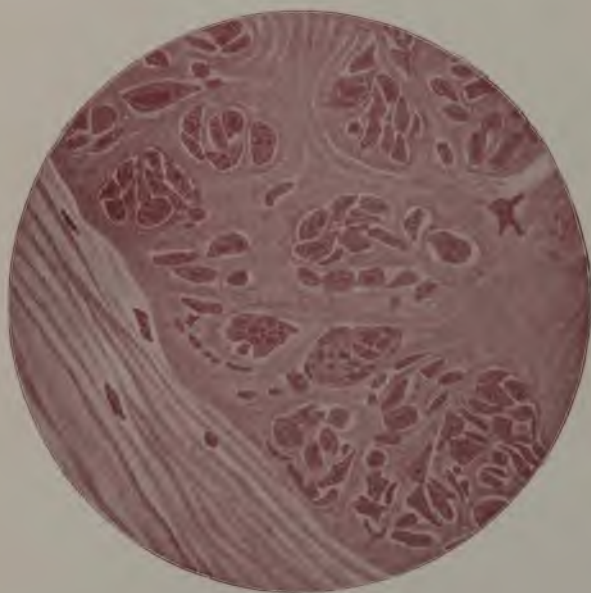


FIG. 4.





# FIVE CASES OF ACUTE LEUKÆMIA

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CASES of acute leukæmia are of sufficient rarity and interest for the following series of cases to be put on record. Two cases were observed by one of us (J. R. B.) as long ago as 1890; the other three have been seen during the past year (1897). Of these three latter cases one was seen by one of us (J. R. B.) in private, in consultation with Dr. Bontor of Berkhamsted. All the other four cases, two in 1890 and two in 1897, were admitted into University College Hospital, some under the care of Dr. Bradford, and some under the care of Dr. Ringer; and we are indebted to Dr. Ringer's courtesy for permission to include his cases in this series.

The nature of the first case in 1890 was detected, one may say, by accident, as a result of an examination of the blood; and the first case being thus recognised the

detection of the other four was easy, as all the cases presented a most remarkable similarity clinically. In three cases a more complete examination of the patient and of the blood was possible; in the remaining two the record is imperfect: in both of these—one a hospital and the other a private case—the patient was only seen when practically moribund, and hence the examination possible was necessarily very imperfect. In all five cases, however, the blood was examined, and in four cases a post-mortem examination was made. This was not obtained in the fifth case.

CASE 1.—Roland C—, aged 30, married, checker on the Midland Railway, was admitted into University College Hospital on September 30th, 1890, complaining of (1) headache; (2) sore throat; (3) tender gums, and (4) pricking pain in the left hypochondrium.

*Past illnesses.*—Patient had rheumatism eight years ago; he has had tonsillitis twice, he has never had scarlet fever, and there is no history of syphilis. Both his parents are alive. He has no children. Patient has lived in London for the last ten years. He states that his home is healthy and his habits are regular. He has always been temperate, drinking one pint of beer a day, but during the past four months he has been practically an abstainer.

*History of present illness.*—He states that for the last three months he has suffered from headache, and during the last month has had to complain of sore throat. A fortnight ago his gums became sore and spongy. During the last month he has complained of great weakness and shortness of breath, but he has been able to go to work until a few days before admission. There is no history of epistaxis.

*Present state.*—Patient is a well-built man of thirty years of age. His skin and mucous membranes are very pale, and he looks intensely anæmic, and the body generally is rather wasted. He can lie in bed in any position.

There is no œdema, the fingers are not clubbed, and the muscles are very flabby. The *alæ nasi* do not work, and there is no dyspnœa at rest. Patient complains of loss of appetite. The tongue is covered with white fur, the breath very offensive; the gums are soft, red, spongy, and bleed readily. There is some pain in the throat, and the tonsils are swollen and spongy-looking. The uvula is large and flaccid. There is no ulceration of the mucous membrane of the gums, tonsils, tongue, or pharynx. The abdomen is rather flat. The upper limit of the liver dulness in the nipple line is at the upper border of the sixth rib, in the middle axillary line at the eighth rib; the lower edge of the liver cannot be felt. The spleen cannot be felt. Both flanks are resonant, the bowels are constipated. The H. A. B. is in the fifth interspace, and the impulse is feeble in character. Heart-sounds are weak, but there is no murmur. Pulse is 80 and regular. There is no cough or dyspnœa. Both sides of the chest move well. Percussion note is good, and the breath-sounds are well heard all over the chest. There are no râles, the vocal fremitus and vocal resonance are normal. The patient has a good deal of pain, starting in the occipital region and passing forward through the head to the frontal region. Sleep, however, is good, and there is no vomiting. The sight is normal, and there is no optic neuritis. The urine is cloudy and deposits urates, sp. gr. 1022; contains a trace of albumen, but no sugar.

October 4th.—A number of bluish-red purpuric spots are noticed on the patient's legs to-day. He still complains of his headache. The blood was examined to-day, and the number of red corpuscles in ten squares was 205, white corpuscles 55, hæmoglobin 40 per cent. (Gowers' hæmocytometer was used).

6th.—The state of the mouth is worse, the gums being more swollen, and hæmorrhage having occurred into them.

13th.—The state of the gums is very bad, and such that the patient is unable to close his mouth. The throat, however, is slightly better. The swelling and soreness of

the gums is such as to prevent any solid food being given. The blood was again examined to-day, and 239 red corpuscles and 25 white corpuscles were found in ten squares.

14th.—Patient has now a diffuse swelling involving the right side of the face, otherwise his condition is much the same. This swelling of the face got steadily worse until October 22nd, when it was found that the inside of the right cheek was greatly swollen, and that there was a considerable amount of sloughing of the mucous membrane lining the cheek and the alveolus of the upper jaw. A quantity of slough was removed, leaving a deep chasm in the cheek, which was swabbed out with 1 in 20 carbolic and dressed with iodoform. Patient is still intensely anæmic, the face is almost white.

On October 26th patient was seen by Mr. Pollard, put under chloroform, and a large quantity of slough removed from the right cheek and alveolus of the jaw. The teeth were exposed down to the fangs, owing to the sloughing of the mucous membrane of the gums. In the course of the afternoon the patient became very collapsed, pulse 120, of very low tension. He recovered, however, under free stimulation, and took his food more freely than previously. He became very steadily weaker and more anæmic, and died on October 31st. The last blood examination was made on October 30th, and showed 1 white corpuscle to 5 red, the number being 108 red in ten squares, and 21 white. The urine throughout the illness was of a pale yellow colour, sp. gr. varying from 1012 to 1025. It always contained a trace of albumen. The highest temperature during the illness was  $102.2^{\circ}$ , and the pulse ranged from 72 to 132.

*Post-mortem examination.*—The right cheek was stained a dark purple colour over an area two inches in diameter. There was a sloughing cavity inside the cheek, and the mucous membrane around this and that covering the alveolus of the upper jaw was greatly swollen and necrosed. The swelling of the gums, although not restricted to the right side of the mouth, was much more marked there than

on the left. There was no swelling or sloughing of the mucous membrane of the left cheek. The thymus gland was 3 inches in length, and  $1\frac{1}{2}$  inches in breadth at its widest point. The right lung was adherent to the chest wall, and firmly so to the diaphragm; otherwise the lungs and pleuræ were quite healthy. The heart was exceedingly pale; no valvular disease was found; muscular substance showed well-marked "tabby cat" striation. The pericardium was normal; the blood was still quite fluid. The liver pale, otherwise normal. The spleen was normal in size. The Malpighian corpuscles were not abnormally prominent. There were no infarcts, and the organ seemed of a good colour and texture. The kidneys were very pale. Ureters and bladder normal, and no calculi or deposits of uric acid were found. The intestines and Peyer's patches were normal. The pancreas was normal. The lymphatic glands all over the body were somewhat enlarged, especially in the neck. The biggest glands, however, were not larger than the thumb-nail. The abdominal glands were very soft and greenish in colour, exuding milky juice on section. The other glands were firmer. Both femora, the left humerus, and the right tibia were examined, and all showed marked changes in the marrow. The shafts of all these long bones were destitute of fatty marrow, and were occupied by soft gelatinous marrow of a red colour, and in places hæmorrhages had occurred into this marrow, and there the colour was dark red.

CASE 2.—William A. H—, aged 58, engineer. Admitted to University College Hospital December 17th, 1890, complaining (1) of excessive weakness, (2) shortness of breath on exertion, (3) swelling and soreness of the gums, so that he was unable to take solid food.

*Past illnesses.*—There is no history of syphilis. Patient had scarlet fever badly when six years of age, but he has had no serious illness since. He has been troubled with slight colds and attacks of lumbago. His

mother is said to have died of "old age;" his father died at forty "from drink." He has four sisters alive and well. He has had three brothers, of whom two are dead, one "from drink." He works in a shed which is very draughty. He has had plenty of good food, and has been a teetotaler for the last four years. He never was a heavy drinker. He has never been out of England.

*History of present illness.*—About five weeks ago he states that he began to feel weak, and that he had a heavy feeling in his chest, and that he seemed to lose power in his limbs, so that on arriving at his work he felt so enfeebled that he had to return home. He went to bed, and has never been able to get about since. His mouth and gums became sore about a fortnight ago. He states that he has wasted considerably for the last five weeks.

The patient on admission was exceedingly weak and prostrate, the skin and mucous membranes being exceedingly pale, and the patient had very considerable dyspnoea on the slightest exertion. The gums were greatly swollen, the lips and teeth were covered with sordes, breath exceedingly foul. The gums were not only swollen, but in places were sloughing and necrotic. No marked swelling of the tonsils and pharynx could be made out. Patient was in an exceedingly weak state, and only a very cursory examination could be made. The lymphatic glands were found slightly enlarged. The spleen and liver could not be felt. Nothing abnormal was detected in the chest. The blood, unfortunately, was not examined until the day before death, when the proportion of white to red was found to be 1 to 10, but no count of the red corpuscles and no estimation of the hæmoglobin were made.

Patient died on December 21st, the highest temperature during the illness being  $100\cdot2^{\circ}$ ; the pulse ranged from 100 to 110; the urine was pale in colour, acid; sp. gr. 1012 to 1018, deposited urates, and contained no albumen or sugar.

*Post-mortem examination.*—The thymus was found to

be persistent; the blood was of a "café au lait" colour. The heart was exceedingly pale; there was no valvular disease. There were, however, petechial hæmorrhages beneath the pericardium. The left pleura was adherent, the right pleura partially adherent. There was no excess of fluid in the pericardial, pleural, or peritoneal cavities. The liver weighed 72 ounces, and was pale and fatty. The kidneys were enlarged, pale, and smooth; the capsule stripped off easily. The right weighed  $6\frac{1}{2}$  ounces, the left  $7\frac{1}{2}$  ounces. The lungs were normal. The spleen was slightly enlarged, and contained no infarcts. The mesenteric and retro-peritoneal glands were slightly enlarged, but appeared normal on section. The cervical, axillary, and inguinal lymphatic glands were also slightly enlarged. The marrow in the shafts of the long bones was red and gelatinous in appearance.

CASE 3.—George D—, 19 years of age, French polisher, admitted to University College Hospital February 22nd, 1897. Patient was admitted complaining of (1) swelling and pain in the face, (2) lumps in the neck, (3) spots on the body, and (4) great weakness. For two or three days previously to January 25th, 1897, patient had not felt well, but was not so ill as to cause any anxiety. On January 25th—that is to say, about four weeks ago—he began to have "toothache" in the upper jaw, and two or three days later he noticed some swelling in his neck, which began on the left side and extended to the right side, and this swelling has gradually increased on both sides of the neck. At this time he came to the Out-patient Department, and was seen by one of us (J. R. B.), and the cervical glands were found to be enlarged, especially on the left side. An examination of the mouth was made, but no swelling of the gums, &c., was noticed. About the end of the first week in February he noticed some swelling on the roof of the mouth, more especially on the left side, and this has since increased. About February 15th his face began to swell, and there was

some puffiness of the eyelids. Three or four days before admission he noticed a number of red spots on his body. The weakness began with the illness, and has been steadily getting worse. He has also had slight headache since the beginning.

*Past illnesses.*—Patient had scarlet fever about eight years ago, but he gives no history of any other illness. There is no one else ill in the house.

*Family history.*—Father and mother alive and well. A brother of the patient died of scarlet fever ten years ago. He has an only sister alive and well. Patient works ten hours a day—the work is light. There is no history of privation, and no history of alcoholism.

*Present state* (February 23rd).—Patient is a youth aged 19. His face is very much swollen, more especially on the left side. He states that blood sometimes comes from the left nostril, and he is unable to breathe through the left nostril. He is intensely pale and is evidently very ill. The mouth is kept half open. The lower lip is swollen, and sordes are present on both lips. The breath is very foul. The conjunctivæ are injected. The tongue is very coated. The teeth are very irregular, and those in the left upper jaw are very loose, and embedded in a large fungating mass involving the gum on both sides of the alveolus and the adjacent part of the roof of the mouth. This mass is soft and ulcerated in two places, and bleeds readily. The gums of the right upper and of both lower jaws are red and spongy. The glands of the neck are enlarged on both sides, and more particularly on the left side beneath the sterno-mastoid and the angle of the jaw. These glands are not tender and in some places are quite separate, but in others appear to run together. Similar glands can be felt in the supra-sternal and both supra-clavicular fossæ, also in both axillæ, and in both groins. Both tonsils are enlarged, especially the left, which reaches the middle line. Patient shows general wasting, and there is a general well-marked pallor. On the front of the trunk and on all four extremities there



are irregularly distributed petechiæ, some darker in colour than others, varying from red to brown. The spots occur as high as the neck, and the back, especially in the upper part, is covered with them. The pulse is rapid, 128 to the minute, regular in force and rhythm, and the tension is somewhat increased. Respiration 28 to the minute. There is nothing abnormal to be seen in either fundus oculi. The knee-jerks are present, and there is no œdema of the legs. The temperature is 101·5°.

*Circulatory system.*—Nothing abnormal beyond the presence of two soft but distinct systolic murmurs heard, one at the apex and the other at the pulmonary cartilage.

*Respiratory system.*—No definite physical signs, but the upper limit of the liver dulness is raised in front. There is dulness to percussion, and the breath-sounds are weak at the base of the right axilla. An occasional rhonchus is heard in both lungs.

*Abdominal system.*—The abdomen is somewhat distended. There is no pain or tenderness. Percussion note is everywhere resonant. The liver and spleen and kidneys are not to be felt.

*Urinary system.*—No symptoms; urine normal in colour, sp. gr. 1025, deposits urates, and there is a trace of albumen.

February 24th.—Three loose stools were passed during the night, each of which contained blood-stained fæces; no clots.

26th.—Temperature rose to 102·6° last night. Diarrhœa is present, with blood in the stools, but less severe to-day. The abdomen is distended and tympanitic. A gland in the right groin is tender, but there are no signs of inflammation in its neighbourhood. During the night the patient noticed a painful spot on the right buttock; near its position the skin is reddened over an area about half an inch in diameter, and near the centre it is raised and darker than elsewhere. Nothing could be made out *per rectum* to account for the hæmorrhage from the bowel. The examination of the blood shows an increase in the

white corpuscles. There are no hæmorrhages into the retinae.

28th.—Temperature reached  $103.8^{\circ}$  to-day. The blood was examined to-day and estimations of the red corpuscles made; one showed 2,225,000 per cubic millimetre, the other 2,500,000. One estimation of hæmoglobin showed 40 per cent., the second 30 per cent.; the proportion of red to white was as 35 to 1. There has been no more blood in the motions. The abdomen is distended, and the purpuric area on the right buttock has increased in size, and the skin over it is broken. The spots on the back are beginning to fade.

March 2nd.—The face is still swollen and the left eyelid puffy, and the sloughy condition of the mucous membrane covering the left upper jaw has increased. On the face two or three areas of ill-defined purpura have developed, somewhat slightly raised above the surrounding surface, something like the patch above noticed on the right buttock.

3rd.—The blood was stained to-day with eosin and methylene blue. A large increase of white corpuscles is well shown; many are stained blue only, and are mononuclear and large. Others have very irregular nuclei, and resemble the ordinary polymorpho-nuclear cells of normal blood. There are numerous cells with well-marked eosinophile granules. There are no nucleated red corpuscles. Comparing the estimate with that of February 28th there is a further increase in the number of white corpuscles present. There has been some nose-bleeding this morning, and the gums tend to bleed rather freely. There are some spots on the conjunctiva of the left eye resembling purpura. It is doubtful whether the pain on pressure on the long bones is greater than normal.

4th.—The examination of blood shows red corpuscles 2,000,000 per cubic millimetre; the proportion of white to red is 1 to 7.

5th.—Patient looks, if possible, paler to-day. The increase in the hæmorrhage from the gums was checked

by an application of perchloride of iron. Patient complained of pain over the lower part of the chest and over the upper part of the abdomen. The purpuric areas on the face have increased in size. Towards evening the patient suddenly became collapsed, and died at 9 p.m. The patient was febrile throughout his illness; highest temperature  $103.8^{\circ}$ . He was sick on two occasions, and his urine constantly contained albumen.

*Post-mortem* (seventeen hours after death).—*Head*.—There was no hæmorrhage into the meninges, but small hæmorrhages were seen in the brain substance on section, and also in the choroid plexuses.

*Chest*.—Thymus persistent, measured three inches in length. Petechiæ were present on the heart, especially near the apex, in front of the right ventricle, on the parietal pericardium, on the base of both lungs, and on the parietal pleura, also under the endocardium of the right auricle and left ventricle. The heart weighed 10 ounces. Its muscular substance was healthy-looking and not very soft. The left ventricle not dilated, aortic valves somewhat fibrosed, and two of them were fused together. The mitral valves showed some minute vegetations, especially on the auricular aspect, and the valves were somewhat thickened; the other valves were normal. There was slight atheroma at the commencement of the aorta. The lungs were crepitant throughout, and there was no hæmorrhage into them. A little rather recent lymph was present at the posterior border of the right lung, and at the bases of both lungs. At the bifurcation of the trachea there was a mass of large glands. On section of the glands the surface was seen to be mottled yellow and dark red, and there were some cretaceous masses also cut across. No free fluid in either side of the chest, no tubercle in the lungs.

*Abdomen*.—There was recent lymph in the peritoneal cavity, and also some yellowish fluid, especially in the pelvis. The lymph was most marked in the right flank.

Petechiæ were present all over the peritoneum, but more especially under the peritoneum covering the intestines.

The intestines were distended, and hæmorrhages were seen, both wide-spread and small, under the mucous membrane. In the middle of the small intestine the mucous membrane was raised into numerous elevations of the size of large peas, the summit of each being completely covered with mucous membrane, and when incised seemed to be composed of soft tissue darkly blood-stained. Throughout the intestinal tract, and especially in the stomach, there were numerous small whitish nodules shining through the mucous membrane. In many places the wall of the gut was much pigmented, and this was especially the case in the Peyer's patches.

The mesenteric and other lymphatic glands of the abdomen and pelvis were greatly enlarged, and were dark in colour. On section the cut surface was mottled dark red and yellow. The substance of the glands was very soft and pulpy. The spleen was enlarged, weighing 20 ounces. It did not reach below the costal margin. It was reddish in colour and a little tougher than natural. There were traces of small whitish deposits in it.

The liver weighed 4 lbs. On section the cut surface was light red in colour, and the organ was infiltrated with an immense number of minute whitish deposits about the size of a pin's head.

Both kidneys were enlarged; the capsule stripped off easily. The cortex was pale, and the right kidney weighed 8 ounces. On the left side there was an extensive hæmorrhage under the epithelium of the right renal pelvis. The bladder contained several ounces of urine, free from blood but containing albumen; there were some petechiæ on its inner surface. Parts of both femora were removed; the marrow in each looked red, and in places resembled red-currant jelly. The sternum and ribs on section showed the marrow rather paler than normal. There was an extravasation of blood into the right rectus abdominis, where it lay over the right lower

ribs, and a patch of recent blood extravasation was seen under the skin of the left thigh. The glands of the groin and axilla were dissected, and were found to be free from hæmorrhage, and contrasted with the mesenteric glands. The cervical glands were also free from hæmorrhage.

CASE 4.—Albert M—, aged 7 years. Admitted March 15th, 1897; died March 23rd, 1897. Patient was admitted with following symptoms:

- (1) Swelling of the face (nose and lip) and neck.
- (2) Loss of appetite and exhaustion.
- (3) Pain in the right side.

March 15th, 1897.—Previous to the beginning of February, 1897, patient was quite well; he then complained of pain in the right side, and felt very hot and feverish. The doctor called in said that the patient had "inflammation of the lungs." Patient was kept in bed for three weeks, and got a little better and was allowed to get up, but he was then found to be very weak, and coughed a good deal; cough was dry and hacking in character, and patient is said by his mother to have coughed up small lumps of material like "pale blood." His appetite was noticed to be poor.

At the end of February patient was brought to this hospital. The pain in the side was much better, but the cough was still present, and the weakness had increased. At this time there was no very marked swelling in the neck and face, but some glandular enlargement was noticed on both sides of the neck. The mother was given some ointment to rub into the neck and some medicine to be given internally.

On March 4th the patient was again brought to the hospital, *i. e.* about a week since last visit. The enlargement of the glands in the neck was less, the cough was better, nor was there any pain in the side; but the weakness had greatly increased, so marked was it that patient could not walk from his home to the hospital.

11th.—Mother again brought the child to the hospital, as he had become much worse—losing flesh and perspiring very freely at night while asleep. His breath, which had become offensive lately, was now much more so.

On March 14th, the child's face was first noticed to be swollen, especially the lips and nose. The child was admitted into the hospital on the 15th March, 1897.

Ever since the beginning of February patient has been getting thinner and paler. Previous to this he was a rosy-cheeked well-nourished child. He has never been sick; his bowels have been rather constipated, more at times than others.

*Past illnesses.*—Measles in 1895, whooping-cough in 1896. No scarlet fever or other illness beyond bad colds.

*Family history.*—Mother, aged 41, alive and fairly well. She has had eight miscarriages—eight full-time children, and only four of latter are alive: it is doubtful whether the other four died of consumption. Father, aged 38, alive, not strong. His mother died of consumption. Patient is the youngest child but one alive; the other three are weakly.

*General history.*—House clean and dry. The children, however, have ailed since they went to live at Pentonville.

*Present state.*—Patient is an ill-nourished spare child of 7 years. He is very pale, and the sclerotics look very blue. There are purpuric spots on the body, more especially at the back and upper part of the chest, and in front on the lower part of the neck, and slightly on the right side of the neck, also on the front of the legs, more especially on the left thigh, and on both aspects of the arms, especially the left. Patient sweats rather freely, and the skin of the body is smooth and moist; the veins of the hands show through the skin; there is no œdema anywhere, and no pain on percussion of the bones; knee-jerks present. Two slight abrasions of the skin are present on the right side of the face near the eye (see photo-

graph, Pl. XI), with a certain amount of pigmentation as if due to blood extravasated beneath.

*Mouth.*—There is a sanious discharge from the nostrils, and swelling and redness around the left nostril. The upper lip is swollen and lifted off the upper jaw. The mucous membrane over the swollen part is ulcerated for about half a square inch. The mucous membranes generally are very pale. The incisor teeth of the *lower* jaw are rather loose. There is a good deal of swelling of the mucous membrane covering the right upper jaw, involving also the hard palate; there is some hæmorrhage from the right upper gum, slight ulceration being present. The gum of the right lower jaw is also swollen, and bleeds a little. The breath is very offensive.

There is a definite glandular enlargement in the neck, none in the axillæ or groins; the largest gland in the latter position is not larger than a haricot bean.

In the neck the glands are enlarged in both posterior triangles, near the angles of the jaw, below the lobules of the ears, along the anterior border of the sterno-mastoids, and beneath the body of the lower jaw. The greatest glandular enlargement is on the right side of the neck, and the largest gland is almost as big as a pigeon's egg, and is just below the right lower jaw. The glands are moveable, separate from one another, and on left side rather tender.

The appetite is fairly good, but it is painful to patient to take his food. Bowels regular. No purpura under conjunctivæ. Radial pulse is fairly compressible—160 to the minute, regular in force and rhythm. Respiration 24, temperature 102·8°. Pupils are widely dilated, equal, and react to light and accommodation. Movements of eye-balls normal.

*Circulatory system.*—The vessels of the neck pulsate markedly from clavicles to lobules of ears. External jugular veins are not distended. The cardiac impulse is somewhat diffuse in the third, fourth, and fifth spaces to the left side of sternum, internal to the nipple line. There

is no marked enlargement of heart; an apical systolic murmur and a systolic murmur at the pulmonary cartilage, where the second sound is accentuated, are heard.

*Respiratory system.*—Patient has a slight cough. There are no signs except numerous râles and rhonchi on both sides of the chest, and these are especially marked at the bases.

*Abdominal system.*—No symptoms. Abdomen is slightly distended, but moves well on respiration; no mass to be felt, but there is some resistance below the right costal margin down to the level of the umbilicus. Edge of liver not definable. The spleen can be felt a finger's breadth below left costal margin. Dulness to percussion is noticeable below right costal margin to within a finger's breadth of umbilicus. There is some pain on deep palpation of the right iliac region.

*Urinary system.*—No albumen, no blood.

March 15th.—Blood examination: hæmoglobin, 36 per cent.; red corpuscles, 1,480,000 per c.mm.; white to red, 1 in 43. By staining there was found a great increase in the number of large mononuclear cells.

17th.—Upper lip was swollen; motion to-day quite free from blood, but very offensive.

18th.—There was a good deal of fever; patient's upper lip has become much more enlarged, and the surface of the mucous membrane is very excoriated. Gums as before. Tonsils are pale and not enlarged. The glands in the neck are more tender than on admission. No hæmorrhages were seen in retinae. A dark and deeply seated purpuric spot has developed over right buttock. Other purpuric spots are unaltered. Pulse getting more frequent. Blood examination: hæmoglobin, not examined; red corpuscles, 45 per cent.; white to red, 1 in 33.

19th.—Two sore places have been noticed on the back of the head to-day near occiput. There is tenderness over them, and they are raised above the skin. It is very difficult to feed the patient, as he complains so much during the feeding, not at other times.



21st.—Sore places on back of head are more tender and more extensive. The two patches noticed on right side of face are more pronounced. Over the right wrist a papule, first noticed on March 15th, has now become almost as big as a pea. The upper lip is still swollen, and its surface covered with a brownish-black scab. There is a purulent discharge from the nostrils. Deep-seated purpuric patches have developed on the legs. The patient complains of pain in the abdomen. The spleen is not so much enlarged as it was. The pain in the abdomen cannot be localised, nor is it increased by pressure. A motion passed this morning looked tar-like in colour and consistency. Patient is difficult to feed, and he is very fretful; much œdema about right eye.

22nd.—Blood examination: hæmoglobin, 26 per cent.; red corpuscles, 30 per cent.; 1 white to 25 red.

23rd.—The two patches to the right of the right eye are much larger to-day, and show more red colour; they are scaly on the surface. A cultivation was attempted from the blood to-day (no growth was obtained by Dr. Curtis). Breath is still very offensive. Right upper gum is less swollen than it was, and paler. Patient's skin looks very waxy, and the veins show through very distinctly as pink lines. Raised area over right wrist is more extensive. Bowels confined. Pulse and respiration are 154 and 48 to the minute. Patient became gradually weaker during his stay in hospital, and rapidly sank and died to-day. Temperature on March 17th and 18th reached  $105\cdot4^{\circ}$ ; at death it was  $100^{\circ}$ . A trace of albumen appeared in the urine towards the last.

*Autopsy* (March 24th, seventeen hours after death).—Rigor mortis present in slight amount.

*Head*.—Brain very pale; no hæmorrhages into it or into the meninges; 2 or 3 drachms of clear fluid in cranium after removal of brain. A portion of the gum of the right upper jaw was removed and placed in alcohol. It was not ulcerated, nor did the bone seem necrosed. One of the enlarged glands on the right side of the neck

was removed, and on section was pale, with some reddish coloration. No tubercle present, and centre not softened.

*Chest.*—Thymus present, not particularly enlarged. It was very pale, and was separated with difficulty from surrounding tissues. Pericardium contained a couple of drachms of straw-coloured fluid. Petechiæ present on right auricle and right ventricle. All the valves were normal except the aortic, which was a little thickened; the right lung was adherent at the apex. Both lungs showed emphysema, collapse, and broncho-pneumonia. There was purulent (?) secretion in many bronchioles. Glands in the posterior mediastinum only slightly enlarged, but redder than normal.

*Abdomen.*—Liver weighed 1 lb. 12 oz. ; it was fatty and free from adenoid growth.

*Spleen* weighed  $4\frac{1}{2}$  ounces. On section the surface was somewhat mottled, reddish and purple. Spleen rather soft, and on surface at one spot there was a yellowish deposit. On cutting through this the outline was irregularly conical, and the deposit was sharply marked off from the spleen substance.

*Kidneys.*—Each kidney weighed  $4\frac{1}{2}$  ounces. They were pale, and scattered through the cortex were definite deposits, and in one of these deposits there was some hæmorrhage.

There were a few petechiæ in the omentum and peritoneum.

The glands of the mesentery were not enlarged, but some of them were redder than normal.

*Intestines.*—There was a small hæmorrhage in one part of the small intestines. In the duodenum there were four places where the mucous membrane had been raised by gas (probably due to decomposition). There was a similar appearance of emphysema in one of the mesenteric glands.

The lower part of the large intestine was dotted over with rather pale elevations with little black centres, as if due to old hæmorrhages into them.

*Marrow.*—In the femur the marrow was of the colour of fuller's-earth ; in no place was there an appearance of red-currant jelly. The marrow of the sternum was pale brown, as was also the case in the ribs.

An occasional small hæmorrhage was found in the muscles of the chest wall.

Nothing was found to account for the tarry motions. Towards the lower end of the ileum, near the ileo-cæcal valve, there seemed to be some slight increase in the lymphoid tissue of the solitary follicles, but there was no definite heaping up. Almost throughout the small intestines the Peyer's patches were darker than normal, but not at all prominent.

CASE 5.—W. G—, aged 17, schoolboy, was seen by one of us (J. R. B.) in December 16th, 1897, in consultation with Dr. Bontor of Berkhamsted.

Patient, although always delicate, has had no previous serious illness, and has been quite well until about five weeks previously.

The only point of interest in his past history was that he disliked most fresh vegetables, and had got into the habit of rarely if ever eating any. Five weeks before seeing him patient had been taken ill with vague symptoms of general malaise and weakness, but he recovered sufficiently to come home from school in about a fortnight from the onset. At this time he was sufficiently well to be up and about, but was noticed to be very weak and extraordinarily pale. He improved somewhat under treatment, and was able to go out, but very soon the weakness and pallor returned in an aggravated form, and patient was confined to his bed. About a week before he was seen by one of us the gums began to swell. He had no nose-bleeding, nor hæmorrhage from any mucous surface. When seen the patient was extraordinarily pale ; the mouth was half open, the gums were swollen to such an extent that the teeth were lying in a furrow. The breath was very offensive, and the gums in places were

sloughing. There was considerable enlargement of the cervical glands, and the glands in the left groin were also slightly enlarged, but were not very tender; the bones, on the other hand, were exceedingly tender. The abdomen was distended. The tip of the spleen could just be felt below the costal margin; the liver was not felt. There was very great dyspnoea, and the pulse was rapid and exceedingly weak. Further examination of the blood showed that there was great excess of white corpuscles, but a differential count could not be made, as no hæmocytometer was available. The films were, however, stained, and the results are given below. The patient died the same night, and a post-mortem examination was not obtained.

We shall use the following nomenclature in describing the variety of white cells present in the blood in these cases :

(1) Lymphocyte—large or small. The small lymphocytes stain deeply, and have a round nucleus concentrically situated in the cell. These small lymphocytes are from 5 to 6  $\mu$  in diameter.

(2) The large lymphocytes. These stain faintly; the nucleus is of variable shape, generally central in position; the protoplasm is more abundant than in the small variety. The size of the cell is variable, but we have found some reaching from 16 to 18  $\mu$ .

(3) Polymorpho-nuclear cells. This is the most convenient term by which to describe the ordinary and most abundant white corpuscle of normal blood. The nucleus is partite, irregularly stained, and the cells contain oxyphile granules, always small.

(4) Coarse oxyphile cells of varying size, the nucleus single or partite. These cells are readily recognised, as they invariably contain very coarse oxyphile granules.

(5) Myelocyte. The nucleus stains faintly with methylene blue, and it may be round, oval, or lobed, and is situated at one pole of the cell, and the contour of the nucleus

is in close contact with the cell wall over a portion of its extent. The granules are usually oxyphile and small. The size of the cell is variable, but they are met with up to 20  $\mu$  in diameter.

*Description of the blood.*<sup>1</sup>

Case 3.—George D—. Proportion of white to red varied between 1 to 7 and 1 to 35. No nucleated red corpuscles were found. The highest number of red corpuscles observed was 2,500,000, the lowest number observed 2,000,000; white corpuscles 74,000 to 280,000. Percentage of different kinds of corpuscles: small lymphocyte, 6·9 per cent.; large lymphocyte, 90·4 per cent.; polymorpho-nuclear, ·5 per cent.; coarse oxyphile, 1·6 per cent.; myelocyte, ·6 per cent.

Total number of white corpuscles counted, 4000. (Plate X, fig. 1.)

Case 4.—Albert M—. Proportion of white to red varied from 1 to 25 to 1 to 43. No nucleated red corpuscles were seen. Highest number of red corpuscles 2,500,000, lowest 1,500,000. Number of white corpuscles varied from 34,500 to 68,000 per c.mm.; small lymphocytes, 12·2 per cent. and 13·4 per cent.; large lymphocytes, 61·8 per cent. and 64·3 per cent.; polymorpho-nuclear and coarse oxyphile, 26 per cent. and 22·3 per cent.; myelocyte 0 per cent.

*Note.*—The coarse oxyphile cells were very scarce, but were present. No myelocytes were seen, with one doubtful exception. Total number of white corpuscles counted, 1402. (Plate X, fig. 2.)

Case 5.—W. G—. Great excess of white corpuscles present, but no count made, as no hæmocytometer was available. Small lymphocytes, ·88 per cent.; large lymphocytes, 98·49 per cent.; polymorpho-nuclear, none found; coarse oxyphile, ·23 per cent.; myelocytes, ·4 per cent. (Plate X, fig. 3.)

<sup>1</sup> It will be remembered that in Cases 1 and 2 stained films were not made.

Films from the marrow in the shaft of the femur were made in Cases 3 and 4; the differential counts yielded the following results:

Case 3.—Small lymphocytes, 3·3 per cent.; large lymphocytes, 92·6 per cent.; polymorpho-nuclear, none found; coarse oxyphile, none found; myelocytes, 4·1 per cent.

Total number of corpuscles counted, 832.

Case 4.—Small lymphocytes, 16·8 per cent.; large lymphocytes, 82·2 per cent.; polymorpho-nuclear, 0·2 per cent.; coarse oxyphile, 0·6 per cent.; myelocytes, 0·2 per cent.

The blood examination in all five cases shows that they were all undoubtedly cases of leukæmia. It is unfortunate that in the first two cases stained blood films were not made, so that we are unable to say whether the leukæmia in these cases was dependent upon an excess of lymphocytes, large or small, or whether the leukæmia was a mixed one. The fact, however, that in case No. 1 the number of white cells reached the proportion of one white to less than four red, and in case No. 2 one white to ten red, shows that these were undoubted cases of leukæmia, and not cases of some secondary form of leucocytosis.

All the cases came under observation for anæmia or for gangrenous stomatitis, but in all the five cases there was distinct evidence that the stomatitis was not an initial symptom. In case No. 2 the state of the patient precluded a satisfactory history, but it is clear that weakness preceded the stomatitis. In case No. 1 the gums are merely noted as "spongy" when the patient was first seen, and there was then no sloughing of the gum; the patient, however, was extremely weak and intensely pallid. In case No. 3 one of us (J. R. B.) saw the patient in the Out-patient Department of University College Hospital some four weeks before he was admitted, and examined the mouth specially in order to determine,

if possible, the cause of the enlargement of the glands of the left side of the neck, but nothing abnormal in the mouth was detected at this period. This patient came to the Out-patient Department complaining of weakness and of the enlargement of the cervical glands, and he was even then very pale.

In case No. 4 the history is definite that the child was ill previously to the onset of the stomatitis, and in the fifth case Dr. Bontor examined the mouth at the commencement of the illness, and found nothing abnormal until the last week of the patient's life. It is therefore evident that although finally the stomatitis was the most prominent clinical feature apart from the intense anæmia, yet this stomatitis was not the initial lesion, although without a blood examination it would have been easy to mistake these cases clinically for cases of stomatitis. The degree of anæmia was very remarkable, and, in fact, it was so intense and peculiar that the nature of the cases could be recognised almost by this sign alone. The illness in all five cases was accompanied by fever, and in No. 4 this was considerable, reaching  $105.4^{\circ}$ ; in all the other cases the temperature rarely exceeded or reached  $102^{\circ}$ . In No. 3 the teeth were found at the post-mortem to be loose; the cause of this was not satisfactorily determined.

The degree of leukæmia varied in the four cases where the amount could be determined, the extremes being 1 to less than 4 red in case No. 1, and 1 to 43 red in case No. 4. In No. 5 the excess was very great, but no hæmocytometer was available. To the eye it appeared so great that the number of white corpuscles seemed almost to equal that of the red. The nature of the leukæmia in all the cases where the blood was stained showed that the excess of white cells was almost entirely dependent on the presence of the large variety of lymphocytes. In none of the cases where stained films were made was there any notable increase in the ordinary polymorpho-nuclear leucocyte, such as occurs in any secondary leucocytosis; and in

two out of the three cases examined (Nos. 3 and 5) these cells were extremely scanty. In the fourth case they only amounted to 26 per cent. In the third and fifth cases, in addition to a great excess of large lymphocytes, there were a few myelocytes, but nothing comparable to the increase seen in ordinary spleno-myelogenous leukæmia. In all cases there was not only a great diminution in the number of red corpuscles, but there was also a great diminution in the amount of blood, as it was often difficult to obtain blood freely on puncturing the finger. On post-mortem examination, although the bulky clots described by some authors in ordinary chronic leukæmia were not obvious, yet the "café au lait" colour of the blood was very striking. The stomatitis was extreme in amount, and the sloughing and gangrene of the gums, palate, and sometimes of the cheek and lips caused excessive fœtor of the breath. In one case (No. 5) the swelling of the gums was so extreme as to cause the teeth to lie in a furrow; in others the swelling and gangrene were localised, and more especially affected the parietal mucous membrane of the inside of the cheek or lips, leading in two cases (Nos. 1 and 3) to so much swelling of the cheek that a growth of the superior maxilla was at one time suggested as a possible diagnosis before the blood examination revealed the nature of the cases.

In all the cases examined post mortem it was seen that, however extensive the swelling and gangrene of the soft parts of the gums, lips, and cheeks had been, there was no obvious disease of the periosteum or of the bones.

Stomatitis is also occasionally seen in the other and more common chronic form of leukæmia, and depends probably upon the leukæmic infiltration of the gums, &c., with subsequent hæmorrhage into the morbid tissue, and in this way the sloughs and gangrene are produced. The lesions in the gums are very similar in their minute anatomy to the lesions found in one of the cases (No. 3) in the intestines and in the brain. In all the cases there was some enlargement of the superficial lymphatic glands,



and in all cases examined post mortem the abdominal glands, mesenteric and retro-peritoneal, were enlarged except in case No. 4. In three cases (Nos. 3, 4, and 5) the enlargement of the superficial glands was obvious, but in only one (No. 3) was it very marked. In this case undoubtedly the enlargement of the lymphatic glands and of the lymphoid tissue of the intestine was the most prominent feature of the morbid anatomy. In two cases (Nos. 1 and 3) the enlarged abdominal lymphatic glands were soft, and in places almost diffuent. The other glands in these two cases and all the glands in the other two cases examined post mortem were firm on section. In the only case (No. 5) not examined post mortem the accessible glands felt firm, and were but slightly enlarged, except the cervical glands, which were considerably enlarged. In two cases only (Nos. 3 and 5) could the spleen be felt during life, and then only to the extent of a couple of fingers below the costal margin. In two cases no enlargement was found after death. In the other two there was some enlargement; but even in No. 3, where the maximum enlargement occurred, the organ weighed but 20 ounces. In all the cases examined post mortem the thymus was found present or enlarged. The marrow of the long bones was in all the cases diseased; in three (Nos. 1, 2, and 3) it was red and jelly-like in consistence, and in No. 4 it had a puriform aspect, and in no case was the normal fatty marrow present in the shafts of the long bones. Clinically some of the cases presented tenderness of the bones, and in one case (No. 5) this was a very marked feature. The purpuric hæmorrhages in the skin and in the serous membranes to a slighter extent were present in four out of five of the cases, and melæna was present in at least two cases (Nos. 3 and 4). In No. 3 it was occasionally marked, and in this case it may have arisen from the hæmorrhages into the hypertrophied lymphoid tissue of the intestine, leading to the formation of small hæmorrhagic ulcers, although no such ulcer was found post mortem. In all cases the illness was very

acute, and the duration did not exceed eight weeks in any case. Death resulted from asthenia from the intense anæmia, and was not caused by inflammation of the serous membranes or viscera. In the only case (No. 3) where there were signs of serous inflammation in the pleura and peritoneum this was very slight in amount, and in No. 4 there was some broncho-pneumonia.

These cases resemble those described in 1895 by Fraenkel,<sup>1</sup> although in many of his cases described under the term "acute" the duration was longer than six weeks. It is perhaps doubtful whether these cases should be described as cases of acute lymphatic leukæmia, since in some of them this nomenclature is liable to cause confusion.

Lymphatic leukæmia may mean one of two things: (1) a variety of leukæmia where the main post-mortem lesion is enlargement of the lymphatic glands or other lymphatic tissue; (2) a variety of leukæmia in which the excess of white cells in the blood consists largely of lymphocytes, large or small. It is very probable, but not absolutely certain, that these two conditions are really one and the same; but certainly some cases of leukæmia not reported in this series, and characterised clinically by great splenic enlargement, show that the leukæmic condition of the blood is dependent mainly upon the presence of lymphocytes. Further, in Cases 3 and 4, where there was most lymphatic enlargement, the blood condition was not one showing a simple increase of lymphocytes, although these formed the bulk of the white cells present; there were in addition a few myelocytes, doubtless associated with the disease of the bone marrow. It is only right to say that in cases described as "acute lymphatic leukæmia" by other authors, myelocytes were present to a small extent in the blood, as in our cases. Hence we think it better to describe these cases simply as cases of acute leukæmia, although in many respects, and especially in the great

<sup>1</sup> Fraenkel, 'Deutsche medicinische Wochenschrift,' 1895, pp. 639, 663, 676, 699, 712. Other references to the subject will be found in this paper.

abundance of the large lymphocytes in the blood, they resemble and are probably identical with the cases described by other authors as "acute lymphatic leukæmia." However, the first case, at any rate, might, from the post-mortem lesions, be fairly described as a case of pure myelogenous leukæmia, since the spleen was normal in size; and although the lymphatic glands all over the body were enlarged and soft, yet the biggest was but the size of the thumb-nail. On the other hand, the medulla of all the long bones examined was diseased, and there was a very great increase in the white cells of the blood in this case; but unfortunately no stained films were made, so there is no evidence to say what variety of cell predominated, whether lymphocyte or myelocyte. It is remarkable that in Case 4, where the lymphatic glands were not excessively enlarged, except in the neck, where they were moderately enlarged, and the spleen was but slightly enlarged, the excess of white cells in the blood should have consisted almost entirely of lymphocytes. The marrow in this case was excessively diseased, and of the type that has been described by some authors as puriform, and this marrow contained very large numbers of lymphocytes.

Clinically these cases presented a superficial resemblance to certain other diseases, and more especially to purpura and to scurvy. They resembled these maladies in the presence of purpuric hæmorrhages, of sponginess, necrosis, and ulceration of the gums; and in one case (No. 5) there was a history of the patient having avoided vegetables. They were, however, very sharply marked off from these two maladies by the blood examination, as both the number and the character of the leucocytes were quite incompatible with any secondary blood change dependent on anæmia. The blood examination also separated the cases at once from any septic infection arising from a primary stomatitis, since such a leucocytosis would be dependent upon the presence of polymorpho-nuclear cells. These, however, as mentioned above, were always

rare and sometimes absent, the leukæmia being dependent upon the presence of lymphocytes. Further, as mentioned in the clinical record, the stomatitis was a terminal and not an initial symptom. The clinical resemblance to one another of all the cases described by us was so great that we think there can be no doubt that they form a definite and separate group.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 147.)

## TABLE OF CASES.

Case.	Age.	Sex.	Duration of illness.	Purpura.	Melæna.	Tender-ness of bones.	Con-dition of thymus.	Condition of gums and mouth.	Condition of marrow.	Condition of spleen.
No. 1	30	M.	6-8 weeks	Yes	Not ob-served	Not ob-served	3½ × 1½ inches	Stomatitis present, coming on 2 weeks after beginning of illness	Hæmor-rhagic and red and gelatinous	Could not be felt during life; not en-larged P.M.
No. 2	58	M.	5-6 weeks	Yes	Not ob-served	Not ob-served	Pre-sent	Stomatitis present, coming on 3 weeks after beginning of illness	Red and gelatinous	Could not be felt during life; slightly increased P.M.
No. 3	19	M.	5½ weeks	Yes	Yes	No	3 inches long	Stomatitis found, coming on 2 weeks after beginning of illness	Red and gelatinous; micro-scopically many large lympho-cytes and some myelo-cytes	Could not be felt during life; weighed 20 oz. at autopsy
No. 4	7	M.	7 weeks	Yes	Yes	No	Pre-sent, not en-larged	Stomatitis present, coming on 5-6 weeks after beginning of illness	Puriform, not red anywhere; micro-scopically large lymphocytes increased, but only few myelocytes	Could be just felt during life; weighed 4½ oz.
No. 5	17	M.	5 weeks	None ob-served	No	Yes	—	Stomatitis present, coming on 4 weeks after beginning of illness	—	Could just be felt during life

tion ds.	Blood.								Nucleated corpuscles.	Albumen in urine.	Maximum temperature.
	No. of red per c.mm.	Hb.	No. of white to red.	Small lymphocytes.	Large lymphocytes.	Myelocytes.	Coarse oxyphils.	Poly-morpho-nuclear.			
rsally ged, it ially eck	(1) 2,050,000	40%	1 : 3·70		Not	observed			—	Trace	102·2°
	(2) 2,390,000		1 : 10								
	(3) 1,050,000		1 : 5								
rsally itly ged	—	—	1 : 10		Not	observed			—	Nil	100·2°
ficial ids ged, illy in also as in men orax. te its in ines rain	(1) 2,000,000	30% to 40%	(1) 1 : 35	Re- duced	Great- ly mul- tiplied	Pre- sent •	Pre- sent	Great- ly re- duced	None found	Trace	103·8°
	(2) 2,500,000		(2) 1 : 7								
the ical p of ficial ids ged; p ly at l	(1) 1,500,000	36%	1 : 43	Nor- mal or re- duced	Great- ly mul- tiplied	Ab- sent	Pre- sent	Pre- sent, but re- duced	None found	Trace	105·4°
	(2) 2,500,000	26%	1 : 35 1 : 25								
ical ids ially ged; me in roin	—	—	—	Re- duced	Great- ly mul- tiplied	Pre- sent	Pre- sent	Absent	None found	—	—

## DESCRIPTION OF PLATE X.

On Five Cases of Acute Leukæmia (Dr. J. ROSE BRADFORD and  
Dr. H. BATTY SHAW).

In all cases the blood was stained with eosin and methylene blue,  
 $\frac{1}{12}$  oil immersion was used, and the corpuscles drawn to scale.

In each of the figures—

A = Lymphocyte, large variety.

B = Myelocyte.

C = Lymphocyte, small variety.

D = Coarsely granular oxyphile cell.

E = Polymorpho-nuclear cell.

H = Red corpuscle.

J = Lymphocyte, intermediate in size between A and C.

Fig. 1—Blood film from Case 3.

Fig. 2—Blood film from Case 4.

Fig. 3—Blood film from Case 5.



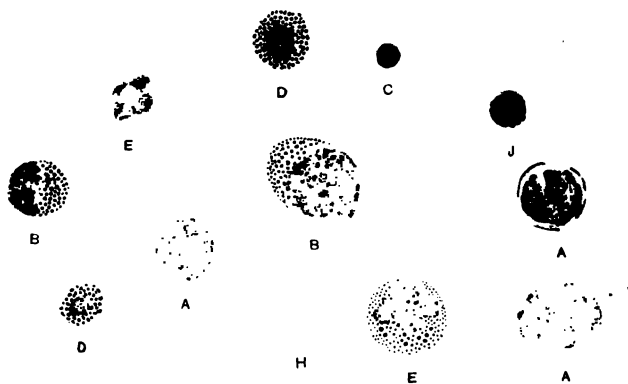


Fig. 1.

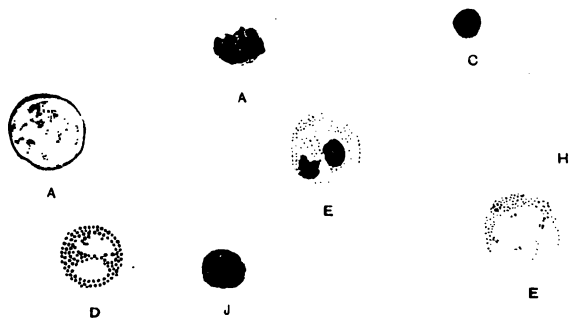


Fig. 2.

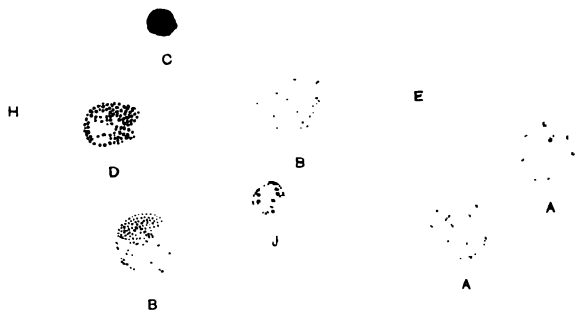


Fig. 3.





**DESCRIPTION OF PLATE XI.**

**On Five Cases of Acute Leukæmia (Dr. J. ROSE BRADFORD and  
Dr. H. BATTY SHAW).**

**Photograph of Case 4, showing the swelling and ulceration of  
the lips.**





# THE INFLUENCE ON GOUT OF THE MINERAL CONSTITUENTS OF VARIOUS VEGETABLES

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IN the Goulstonian Lectures of last year, whilst discussing the relative effects on the gouty process of animal and vegetable diets, I stated that, in my opinion, it was of little importance, as regards the production of uric acid from proteid matter, whether the proteid be of animal or vegetable origin, and that the same harm may result from an excessive consumption of either form of proteid.

But although animal and vegetable proteids may react alike with regard to the production of uric acid, yet it is well known that different articles of diet exercise different influences on the precipitation of sodium biurate, and on the solution and removal of uratic deposits. In connection with this subject, I was able to demonstrate that the saline constituents of animal and vegetable foods exercise very different solvent effects on sodium biurate. From the results of these experiments it appeared probable that if the mineral constituents of vegetables were

present in sufficient quantities in the fluids of a gouty person, they would not only delay the advent of an attack of gout by increasing the solubility of the sodium biurate present in these fluids, but would also by their increased solvent effects on uratic deposits, facilitate the removal of the latter.

Since the delivery of the Goulstonian Lectures I have carried out a long series of experiments with the mineral constituents of all the vegetables in ordinary use, in order to elucidate the two following points :—(1) the relative effects exerted by the mineral constituents of various vegetables on the solubility of sodium biurate at the temperature of the human body; and therefore presumably on uratic deposits; and (2) the influence, if any, exerted by these constituents in retarding the conversion of the sodium quadriurate, which is present in the fluids of the body in gout, into the sodium biurate. Obviously the elucidation of these points would have a material bearing on the treatment of gout.

#### EXPERIMENTAL INQUIRY TO ASCERTAIN THE SOLVENT EFFECTS EXERTED BY THE MINERAL CONSTITUENTS OF VARIOUS VEGETABLES ON SODIUM BIURATE.

The method of carrying out these experiments was as follows :—In each batch of experiments seven bottles, each containing 100 c.c. of distilled water mixed with a known quantity of the vegetable ash, were placed in the warm chamber until their contents were at a temperature of 100° F., when an excess of pure sodium biurate was added to each. The bottles were kept at 100° F. for five hours, during which period they were frequently agitated. At the end of that time the contents of the bottles were filtered, and if necessary re-filtered, through double filters until perfectly clear filtrates were obtained. The amount of uric acid in each of the filtrates was then estimated by adding an excess of strong



sulphuric acid and titrating with a standard potassium permanganate solution; the quantity of uric acid found was subsequently calculated into terms of sodium biurate. The results thus obtained are shown in the following sixteen tables, which are arranged in the order of the average solvent effect exerted by the mineral constituents of the various vegetables, commencing with those exercising the greatest influence.

The solubility of sodium biurate in distilled water is placed at the head of each table for comparison.

TABLE I.—*Showing the influence of the mineral constituents of spinach on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of spinach ash . . . . .	. 3·36 „
0·5 „ „ . . . . .	. 2·76 „
0·2 „ „ . . . . .	. 2·12 „
0·1 „ „ . . . . .	. 1·90 „
0·05 „ „ . . . . .	. 1·52 „
0·02 „ „ . . . . .	. 1·21 „
0·01 „ „ . . . . .	. 1·18 „

TABLE II.—*Showing the influence of the mineral constituents of Brussels sprouts on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of Brussels sprouts ash . . . . .	. 3·06 „
0·5 „ „ . . . . .	. 2·21 „
0·2 „ „ . . . . .	. 1·68 „
0·1 „ „ . . . . .	. 1·62 „
0·05 „ „ . . . . .	. 1·52 „
0·02 „ „ . . . . .	. 1·30 „
0·01 „ „ . . . . .	. 1·23 „

TABLE III.—*Showing the influence of the mineral constituents of potato on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of potato ash . . . . .	. 2·49 ”
0·5 ” ” ” . . . . .	. 2·17 ”
0·2 ” ” ” . . . . .	. 1·92 ”
0·1 ” ” ” . . . . .	. 1·47 ”
0·05 ” ” ” . . . . .	. 1·36 ”
0·02 ” ” ” . . . . .	. 1·12 ”
0·01 ” ” ” . . . . .	. 1·10 ”

TABLE IV.—*Showing the influence of the mineral constituents of asparagus on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of asparagus ash . . . . .	. 2·77 ”
0·5 ” ” ” . . . . .	. 2·09 ”
0·2 ” ” ” . . . . .	. 1·58 ”
0·1 ” ” ” . . . . .	. 1·45 ”
0·05 ” ” ” . . . . .	. 1·33 ”
0·02 ” ” ” . . . . .	. 1·12 ”
0·01 ” ” ” . . . . .	. 1·10 ”

TABLE V.—*Showing the influence of the mineral constituents of Savoy cabbage on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of Savoy cabbage ash . . . . .	. 2·32 ”
0·5 ” ” ” . . . . .	. 1·92 ”
0·2 ” ” ” . . . . .	. 1·77 ”
0·1 ” ” ” . . . . .	. 1·57 ”
0·05 ” ” ” . . . . .	. 1·34 ”
0·02 ” ” ” . . . . .	. 1·13 ”
0·01 ” ” ” . . . . .	. 1·10 ”

TABLE VI.—*Showing the influence of the mineral constituents of French beans on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of French beans ash	. 2·48 "
0·5 " " "	. 1·87 "
0·2 " " "	. 1·68 "
0·1 " " "	. 1·56 "
0·05 " " "	. 1·28 "
0·02 " " "	. 1·16 "
0·01 " " "	. 1·10 "

TABLE VII.—*Showing the influence of the mineral constituents of lettuce on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of lettuce ash	. 2·72 "
0·5 " " "	. 1·92 "
0·2 " " "	. 1·57 "
0·1 " " "	. 1·53 "
0·05 " " "	. 1·21 "
0·02 " " "	. 1·10 "
0·01 " " "	. 1·09 "

TABLE VIII.—*Showing the influence of the mineral constituents of beetroot on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of beetroot ash	. 2·46 "
0·5 " " "	. 1·82 "
0·2 " " "	. 1·60 "
0·1 " " "	. 1·45 "
0·05 " " "	. 1·34 "
0·02 " " "	. 1·15 "
0·01 " " "	. 1·10 "

TABLE IX.—*Showing the influence of the mineral constituents of winter cabbage on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of cabbage ash .	. 2·30 „
0·5 „ „ „ .	. 2·14 „
0·2 „ „ „ .	. 1·63 „
0·1 „ „ „ .	. 1·31 „
0·05 „ „ „ .	. 1·23 „
0·02 „ „ „ .	. 1·10 „
0·01 „ „ „ .	. 1·10 „

TABLE X.—*Showing the influence of the mineral constituents of celery on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of celery ash .	. 2·20 „
0·5 „ „ „ .	. 1·84 „
0·2 „ „ „ .	. 1·53 „
0·1 „ „ „ .	. 1·44 „
0·05 „ „ „ .	. 1·30 „
0·02 „ „ „ .	. 1·10 „
0·01 „ „ „ .	. 1·06 „

TABLE XI.—*Showing the influence of the mineral constituents of turnip-tops on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of turnip-tops ash .	. 2·16 „
0·5 „ „ „ .	. 1·82 „
0·2 „ „ „ .	. 1·58 „
0·1 „ „ „ .	. 1·42 „
0·05 „ „ „ .	. 1·20 „
0·02 „ „ „ .	. 1·13 „
0·01 „ „ „ .	. 1·11 „

TABLE XII.—*Showing the influence of the mineral constituents of turnips on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water	. 1·10 per 1000.
Water containing—	
1·0 per cent. of turnip ash	. 2·04 „
0·5 „ „	. 1·78 „
0·2 „ „	. 1·50 „
0·1 „ „	. 1·42 „
0·05 „ „	. 1·32 „
0·02 „ „	. 1·14 „
0·01 „ „	. 1·10 „

TABLE XIII.—*Showing the influence of the mineral constituents of carrot on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water	. 1·10 per 1000.
Water containing—	
1·0 per cent. of carrot ash	. 1·63 „
0·5 „ „	. 1·53 „
0·2 „ „	. 1·47 „
0·1 „ „	. 1·45 „
0·05 „ „	. 1·33 „
0·02 „ „	. 1·13 „
0·01 „ „	. 1·11 „

TABLE XIV.—*Showing the influence of the mineral constituents of cauliflower on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water	. 1·10 per 1000.
Water containing—	
1·0 per cent. of cauliflower ash	. 1·52 „
0·5 „ „	. 1·50 „
0·2 „ „	. 1·42 „
0·1 „ „	. 1·34 „
0·05 „ „	. 1·28 „
0·02 „ „	. 1·09 „
0·01 „ „	. 1·09 „

TABLE XV.—*Showing the influence of the mineral constituents of sea-kale on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of sea-kale ash .	. 1·49 ”
0·5 ” ” .	. 1·47 ”
0·2 ” ” .	. 1·35 ”
0·1 ” ” .	. 1·23 ”
0·05 ” ” .	. 1·10 ”
0·02 ” ” .	. 1·10 ”
0·01 ” ” .	. 1·10 ”

TABLE XVI.—*Showing the influence of the mineral constituents of green peas on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved.
Water . . . . .	. 1·10 per 1000.
Water containing—	
1·0 per cent. of green peas ash	. 0·99 ”
0·5 ” ”	. 1·01 ”
0·2 ” ”	. 1·04 ”
0·1 ” ”	. 1·10 ”
0·05 ” ”	. 1·10 ”
0·02 ” ”	. 1·10 ”
0·01 ” ”	. 1·10 ”

From the results detailed in these tables it is evident that 0·05 per cent. and over of the mineral constituents of nearly all the vegetables, very appreciably increase the solubility of sodium biurate. The solitary exception is in the case of the mineral constituents of green peas, which practically exert no influence whatever on the solubility of the biurate.

As I considered that these solvent effects of the mineral constituents of most vegetables on the biurate might have an important bearing on the treatment of gout, I next endeavoured to ascertain whether these effects were due to the alkalinity of the vegetable ashes, or whether they could be referred to any one saline constituent of the vegetables.

EXPERIMENTAL PROOF THAT THE SOLVENT EFFECTS OF THE MINERAL CONSTITUENTS OF VEGETABLES ON SODIUM BIURATE ARE NOT DUE TO THEIR DEGREE OF ALKALINITY.

That the solvent effect exerted respectively by the mineral constituents of each vegetable on the sodium biurate was not proportional to the alkalinity of the ash was very easily determined. I made estimations of the alkalinities of the different vegetable ashes, and calculated the percentages of alkalinity in terms of sodium carbonate. The alkalinity of the ashes was due to potassium and sodium carbonates; none of the ashes contained either potassium or sodium hydrate. The following table shows a comparison of the solubility exerted by the mineral constituents of vegetables on sodium biurate, and the alkalinity of those constituents.

TABLE XVII.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the alkalinity of those constituents.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the alkalinity of their ashes, and showing percentages of alkalinity, reckoned as sodium carbonate, commencing with the most alkaline.	
Spinach	...	Spinach . . .	26·00
Brussels sprouts	...	Celery . . .	20·80
Potato	...	Turnip . . .	20·80
Asparagus	...	Potato . . .	17·55
Savoy cabbage	...	Beetroot . . .	15·60
French beans	...	Cauliflower . . .	13·20
Lettuce	...	Carrot . . .	13·00
Beetroot	...	Brussels sprouts . . .	12·35
Cabbage	...	French beans . . .	12·35
Celery	...	Turnip-tops . . .	11·70
Turnip-tops	...	Lettuce . . .	11·05
Turnip	...	Asparagus . . .	8·45
Carrot	...	Cabbage . . .	5·85
Cauliflower	...	Green peas . . .	5·20
Sea-kale	...	Savoy cabbage . . .	4·55
Green peas	...	Sea-kale . . .	1·95

It is evident from a glance at this table that the solvent effect of a vegetable ash on sodium biurate, with the exception of spinach ash, bears no relationship, either of a direct or an inverse ratio, to the alkalinity of the ash. For instance, it can be seen that the solvent effect on the biurate of the ash of Brussels sprouts is high, while its alkalinity is low; on the other hand, the solvent effect on the biurate of the ash of celery is low, while its alkalinity is high.

In other words, it is evident that the order in which the vegetables are arranged as regards the solvent effect of the mineral constituents on the biurate, is neither the order nor the inverse order of their relative alkalinities. These results support the conclusions I arrived at from some experiments made with blood serum, and described in the Goulstonian Lectures of last year. I then showed that a diminution in the alkalinity of blood-serum did not cause a diminution in the solvent power of the serum for biurate, and, conversely, that an increase in the alkalinity of the serum did not increase its solvent power for the biurate.

#### EXPERIMENTAL PROOF THAT THE SOLVENT EFFECTS OF THE MINERAL CONSTITUENTS OF VEGETABLES ON SODIUM BIURATE ARE NOT DUE TO ANY SINGLE CONSTITUENT.

The next problem to solve was whether the effect exerted by the mineral constituents of vegetables in increasing the solubility of sodium biurate is due to any one constituent. With regard to this point it appeared probable beforehand that such would not prove to be the case, since Sir W. Roberts has shown that sodium, calcium, and magnesium salts diminish the solvent power of water on sodium biurate, and that potassium salts exercise no influence, either for or against, on the solubility of the biurate.



Now it can easily be demonstrated that the solvent effect is not due to the potassium salts. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of potassium salts present.

TABLE XVIII.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of potassium salts present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of potassium salts present, and showing the percentages of potassium salts present in the ashes, reckoned as potassium oxide, commencing with those richest in potassium salts.	
Spinach	...	Potato	. . 56·03
Brussels sprouts	...	Turnip	. . 54·05
Potato	...	Carrot	. . 53·36
Asparagus	...	Lettuce	. . 48·01
Savoy cabbage	...	French beans	. . 46·50
French beans	...	Asparagus	. . 39·21
Lettuce	...	Green peas	. . 38·96
Beetroot	...	Beetroot	. . 38·33
Cabbage	...	Cabbage	. . 37·71
Celery	...	Brussels sprouts	. . 35·00
Turnip-tops	...	Celery	. . 33·14
Turnip	...	Turnip-tops	. . 30·55
Carrot	...	Savoy cabbage	. . 26·82
Cauliflower	...	Cauliflower	. . 23·46
Sea-kale	...	Spinach	. . 23·43
Green peas	...	Sea-kale	. . 2·59

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate bears no relationship, either of a direct or an inverse ratio, to the proportions of potassium salts present. For instance, it can be seen that the solvent effect on the biurate of the ash of spinach is high, while the proportion of potassium salts is low; on the other hand, the solvent effect on the biurate of the ash of turnips is low, while the proportion of potassium salts is high.

It can also be demonstrated that the increased solubility of the biurate effected by the mineral constituents of vegetables is not due to the sodium salts. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of sodium salts present.

TABLE XIX.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of sodium salts present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.			Vegetables arranged in order of the proportions of sodium salts present, and showing the percentages of sodium salts present in the ashes, reckoned as sodium oxide, commencing with those richest in sodium salts.		
Spinach	...		Sea-kale	.	33·84
Brussels sprouts	...		Spinach	.	31·42
Potato	...		Beetroot	.	31·17
Asparagus	...		French beans	.	30·50
Savoy cabbage	...		Celery	.	19·33
French beans	...		Asparagus	.	16·79
Lettuce	...		Carrot	.	14·17
Beetroot	...		Savoy cabbage	.	13·86
Cabbage	...		Brussels sprouts	.	12·60
Celery	...		Lettuce	.	11·80
Turnip-tops	...		Cauliflower	.	10·87
Turnip	...		Turnip	.	6·37
Carrot	...		Green peas	.	5·20
Cauliflower	...		Turnip-tops	.	4·19
Sea-kale	...		Cabbage	.	2·39
Green peas	...		Potato	.	2·18

It is evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate bears no relationship, either of a direct or an inverse ratio, to the proportions of sodium salts present. For instance, it can be seen that the solvent effect on the biurate of the ash of potato is high, while the proportion of sodium salts is low; on the other hand, the solvent effect on the biurate

of the ash of sea-kale is low, while the proportion of sodium salts is high.

In like manner it can be demonstrated that the increased solubility of the biurate effected by the mineral constituents of vegetables is not due to the calcium salts. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of calcium salts present.

TABLE XX.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of calcium salts present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of calcium salts present, and showing the percentages of calcium salts present in the ashes, reckoned as calcium oxide, commencing with those richest in calcium salts.	
Spinach	...	Turnip-tops	. 37.15
Brussels sprouts	...	Sea-kale	. 27.56
Potato	...	Cauliflower	. 23.33
Asparagus	...	French beans	. 17.48
Savoy cabbage	...	Cabbage	. 17.14
French beans	...	Lettuce	. 15.02
Lettuce	...	Savoy cabbage	. 14.83
Beetroot	...	Turnip	. 13.38
Cabbage	...	Celery	. 13.06
Celery	...	Spinach	. 10.64
Turnip-tops	...	Carrot	. 6.88
Turnip	...	Brussels sprouts	. 6.16
Carrot	...	Potato	. 5.46
Cauliflower	..	Asparagus	. 5.05
Sea-kale	...	Green peas	. 4.98
Green peas	...	Beetroot	. 2.58

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate bears no relationship, either of a direct or an inverse ratio, to the proportions of calcium salts present. For instance, it can be seen that the solvent effect on the biurate of the ash of potato is high, while the proportion

of calcium salts is low; on the other hand, the solvent effect on the biurate of the ash of sea-kale is low, while the proportion of calcium salts is high.

Similarly it can be shown that the increased solvent effect on the biurate exerted by the mineral constituents of vegetables is not due to either the magnesium or iron salts present.

It can also be demonstrated that the increased solubility of the biurate is not due to the phosphates present in the vegetables. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of phosphates present.

**TABLE XXI.**—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of phosphates present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of phosphates present, and showing the percentages of phosphates present in the ashes, reckoned as phosphoric anhydride, commencing with those richest in phosphates.	
Spinach	...	Green peas	35.62
Brussels sprouts	...	Cauliflower	22.14
Potato	...	Asparagus	21.93
Asparagus	...	Potato	15.99
Savoy cabbage	...	Carrot	15.02
French beans	...	Celery	14.39
Lettuce	...	Brussels sprouts	14.20
Beetroot	...	Savoy cabbage	13.19
Cabbage	...	French beans	12.21
Celery	...	Cabbage	11.99
Turnip-tops	...	Lettuce	9.62
Turnip	...	Turnip	9.26
Carrot	...	Spinach	8.56
Cauliflower	...	Beetroot	8.25
Sea-kale	...	Sea-kale	8.00
Green peas	...	Turnip-tops	6.15

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium bi-

urate bears no relationship, either of a direct or an inverse ratio, to the proportions of phosphates present. For instance, it can be seen that the solvent effect on the biurate of the ash of spinach is high, while the proportion of phosphates is low; on the other hand, the solvent effect on the biurate of the ash of green peas is low, while the proportion of phosphates is high.

It can also be demonstrated that the increased solubility of the biurate is not due to the sulphates present in the vegetables. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of sulphates present.

TABLE XXII.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of sulphates present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of sulphates present, and showing the percentages of sulphates present in the ashes, reckoned as sulphuric anhydride, commencing with those richest in sulphates.	
Spinach	...	Sea-kale	. 19·78
Brussels sprouts	...	Turnip-tops	. 15·27
Potato	...	Cauliflower	. 14·16
Asparagus	...	Savoy cabbage	. 12·85
Savoy cabbage	...	Turnip	. 12·47
French beans	...	Brussels sprouts	. 8·31
Lettuce	...	Cabbage	. 7·28
Beetroot	...	French beans	. 6·82
Cabbage	...	Potato	. 5·60
Celery	...	Asparagus	. 5·40
Turnip-tops	...	Carrot	. 5·20
Turnip	...	Spinach	. 4·44
Carrot	...	Green peas	. 4·36
Cauliflower	...	Lettuce	. 3·92
Sea-kale	...	Beetroot	. 2·41
Green peas	...	Celery	. 1·10

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium bi-

urate bears no relationship, either of a direct or an inverse ratio, to the proportions of sulphates present. For instance, it can be seen that the solvent effect on the biurate of the ash of spinach is high, while the proportion of sulphates is low; on the other hand, the solvent effect on the biurate of the ash of sea-kale is low, while the proportion of sulphates is high.

Finally, as disposing of all the mineral constituents of any importance in vegetables, it can be demonstrated that the increased solubility of the biurate is not due to the chlorides present in the vegetables. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of chlorides present.

TABLE XXIII.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of chlorides present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of chlorides present, and showing the percentages of chlorides present in the ashes, reckoned as chlorine, commencing with those richest in chlorides.	
Spinach	...	Celery	. 22.14
Brussels sprouts	...	Beetroot	. 18.13
Potato	...	Sea-kale	. 15.46
Asparagus	...	Cabbage	. 9.09
Savoy cabbage	...	Lettuce	. 8.80
French beans	...	Spinach	. 7.78
Lettuce	...	Savoy cabbage	. 7.53
Beetroot	...	Turnip-tops	. 7.33
Cabbage	...	Asparagus	. 6.62
Celery	...	Turnip	. 5.06
Turnip-tops	...	Cauliflower	. 4.83
Turnip	...	Carrot	. 3.70
Carrot	...	Brussels sprouts	. 3.00
Cauliflower	...	Potato	. 2.50
Sea-kale	...	French beans	. 2.50
Green peas	...	Green peas	. 2.10

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate

bears no relationship, either of a direct or an inverse ratio, to the proportions of chlorides present. For instance, it can be seen that the solvent effect on the biurate of the ash of Brussels sprouts is high, while the proportion of chlorides is low ; on the other hand, the solvent effect on the biurate of the ash of sea-kale is low, while the proportion of chlorides is high.

These results show that the solvent effect exerted on sodium biurate by the mineral constituents of vegetables is not due to any one constituent.

EXPERIMENTAL PROOF THAT AN ARTIFICIALLY PREPARED  
ASH DOES NOT REACT TO SODIUM BIURATE IN THE  
SAME MANNER AS A NATURAL VEGETABLE ASH.

I next endeavoured to ascertain whether an artificially prepared ash of the same composition as the natural ash of one of the vegetables would exercise a similar effect in increasing the solubility of the sodium biurate as that possessed by the natural ash. For this purpose I selected the spinach ash, which has the greatest solvent effect on the biurate. An artificial ash was prepared which was made with the same proportions of potassium, sodium, calcium, sulphates, phosphates, and chlorides, as those present in the natural spinach ash, and also of precisely the same degree of alkalinity. Experiments were carried out with this artificial ash and the biurate in a similar manner to that employed in working with the natural vegetable ashes. The following table shows the results of these experiments.

TABLE XXIV.—*Showing the influence of the artificial spinach ash on the solubility of sodium biurate at 100° F.*

Solvent.			Sodium biurate dissolved.	
Water	.	.	.	1·10 per 1000.
Water containing—				
1·0 per cent. of artificial spinach ash	.	.	.	0·20 "
0·5 " " "	.	.	.	0·34 "
0·2 " " "	.	.	.	0·62 "
0·1 " " "	.	.	.	0·86 "
0·05 " " "	.	.	.	0·96 "
0·02 " " "	.	.	.	1·04 "
0·01 " " "	.	.	.	1·06 "

These results are very remarkable, as they indicate that the artificial ash exercises in all proportions a deterrent effect on the solubility of the biurate. This deterrent effect is well seen by contrasting the results with those of the natural ash, which show the marked solvent effect exerted by the latter on the biurate.

TABLE XXV.—*Showing the different influences exerted by the artificial and natural spinach ashes on the solvency of the biurate at 100° F.*

Solvent.			Sodium biurate dissolved in 1000 parts.		
Water	.	.	.	.	1·10
Water containing—			Artificial spinach ash.	Natural spinach ash.	
1·0 per cent. of ash	.	.	0·20	...	3·36
0·5 " "	.	.	0·34	...	2·76
0·2 " "	.	.	0·62	...	2·12
0·1 " "	.	.	0·86	...	1·90
0·05 " "	.	.	0·96	...	1·52
0·02 " "	.	.	1·04	...	1·21
0·01 " "	.	.	1·06	...	1·18

The only explanation that I can offer of these remarkable results is that in the natural ash there is some combination of the mineral constituents which cannot be artificially imitated, and that upon this natural combination of the salts is dependent the increased solvent effect



exerted on the biurate by the mineral constituents of most vegetables. If this view be correct, then modern science is but confirming the correctness of the practice of those ancients who employed vegetable ashes in the treatment of gout.

It may be well to make here a brief reference to the experiments that I described in the Goulstonian Lectures of last year, which showed that the mineral constituents of meat exercise a marked deterrent effect on the solubility of sodium biurate, and that this effect is most marked by proportions of the mineral constituents which may certainly be present in the blood after eating a few ounces of meat. The following table shows in contrast the effects exercised respectively by the mineral constituents of lean beef and spinach on the solubility of the biurate.

TABLE XXVI.—*Showing the respective effects exercised by the mineral constituents of beef and spinach on the solubility of sodium biurate at 100° F.*

Solvent.	Sodium biurate dissolved in 1000 parts.		
Water . . .	1·10		
Water containing—	Beef ash.	Spinach ash.	
1·0 per cent. of ash .	0·93	...	3·36
0·5   "       "       " .	0·76	...	2·76
0·2   "       "       " .	0·56	...	2·12
0·1   "       "       " .	0·32	...	1·90
0·05   "       "       " .	0·15	...	1·52
0·02   "       "       " .	0·11	...	1·21
0·01   "       "       " .	0·85	...	1·18

EXPERIMENTAL INQUIRY TO ASCERTAIN THE EFFECT EXERTED BY THE MINERAL CONSTITUENTS OF VARIOUS VEGETABLES ON THE CONVERSION OF SODIUM QUADRIURATE INTO SODIUM BIURATE.

It is well known from the researches of Dr. Bence Jones and of Sir W. Roberts that sodium quadriurate, which is the form in which uric acid first appears in the

blood in gout, is an unstable body, and is gradually converted by combination with the sodium carbonate of the blood into sodium biurate, which latter body, on account of its comparative insolubility, is deposited in the tissues and constitutes the gouty uratic deposit. This gradual conversion of the quadriurate into biurate is known as the *maturation process*.

It is obviously of therapeutical importance to know whether the mineral constituents of any of the vegetables, in addition to exerting an increased solvent effect on the biurate, possess the power of delaying this maturation process; or, in other words, of inhibiting the conversion of the quadriurate into the biurate. In order to ascertain this I conducted a series of experiments. In all these experiments I employed Sir W. Roberts's standard solution, as being a more convenient medium to work with than blood serum. This standard solution contains 0.5 per cent. of sodium chloride and 0.2 per cent. of sodium bicarbonate dissolved in distilled water. Sir W. Roberts found that this solution is a fairly exact representation of blood serum, in so far as its saline ingredients are concerned, and that it reacted with uric acid and the urates in the same manner as blood serum itself, and in the same manner as a solution comprising all the salines of the serum in their due proportions.

The experiments were conducted in the following way: Ten milligrammes of pure sodium quadriurate were well rubbed with ten drops of the standard solution, and the mixture placed in a small corked bottle in the warm chamber and kept at 100° F. Every half hour a small quantity of the mixture was examined under a high power of the microscope, and the time at which crystals of sodium biurate first appeared was noted. This represented the time occupied by the maturation process when the standard solution was saturated with sodium quadriurate. Similar experiments were conducted with the same amount of sodium quadriurate in the same quantity of standard solution, containing respectively 0.1 per cent.

of the mineral constituents of each of the vegetables in ordinary use. The results are shown in the following table:

TABLE XXVII.—*Showing the effects exerted by the mineral constituents of vegetables on the conversion of sodium quadriurate into sodium biurate.*

Solvent.	Crystals of sodium biurate first appeared in
Standard solution . . .	2 hours.
Standard solution containing—	
0.1 per cent. of potato ash . . .	2 „
„ cauliflower ash . . .	2 „
„ lettuce ash . . .	2 „
„ carrot ash . . .	2 „
„ asparagus ash . . .	2½ „
„ beetroot ash . . .	3 „
„ green peas ash . . .	3½ „
„ celery ash . . .	3½ „
„ Brussels sprouts ash . . .	4 „
„ cabbage ash . . .	4 „
„ turnip-tops ash . . .	4 „
„ turnip ash . . .	4 „
„ Savoy cabbage ash . . .	4 „
„ sea-kale ash . . .	4 „
„ French beans ash . . .	4½ „
„ spinach ash . . .	5 „

These results show that the mineral constituents of some of the vegetables, notably spinach, Brussels sprouts, French beans, cabbage, turnip-tops, and turnips, very considerably delay the conversion of sodium quadriurate into sodium biurate. The inference is that if such mineral constituents were present in suitable proportions in the blood of gouty subjects, and if, at the same time, proper measures were adopted for promoting excretion of the quadriurate by the kidneys, the elimination of that body might be secured without the occurrence of any precipitation of the biurate in the tissues. Moreover, it must be borne in mind that these experiments were conducted under very stringent conditions, in that they were all

It can also be demonstrated that the increased solubility of the biurate effected by the mineral constituents of vegetables is not due to the sodium salts. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of sodium salts present.

TABLE XIX.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of sodium salts present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of sodium salts present, and showing the percentages of sodium salts present in the ashes, reckoned as sodium oxide, commencing with those richest in sodium salts.	
Spinach	...	Sea-kale	. 33·84
Brussels sprouts	...	Spinach	. 31·42
Potato	...	Beetroot	. 31·17
Asparagus	...	French beans	. 30·50
Savoy cabbage	...	Celery	. 19·33
French beans	...	Asparagus	. 16·79
Lettuce	...	Carrot	. 14·17
Beetroot	...	Savoy cabbage	. 13·86
Cabbage	...	Brussels sprouts	. 12·60
Celery	...	Lettuce	. 11·80
Turnip-tops	...	Cauliflower	. 10·87
Turnip	...	Turnip	. 6·37
Carrot	...	Green peas	. 5·20
Cauliflower	...	Turnip-tops	. 4·19
Sea-kale	...	Cabbage	. 2·39
Green peas	...	Potato	. 2·18

It is evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate bears no relationship, either of a direct or an inverse ratio, to the proportions of sodium salts present. For instance, it can be seen that the solvent effect on the biurate of the ash of potato is high, while the proportion of sodium salts is low; on the other hand, the solvent effect on the biurate

of the ash of sea-kale is low, while the proportion of sodium salts is high.

In like manner it can be demonstrated that the increased solubility of the biurate effected by the mineral constituents of vegetables is not due to the calcium salts. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of calcium salts present.

TABLE XX.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of calcium salts present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of calcium salts present, and showing the percentages of calcium salts present in the ashes, reckoned as calcium oxide, commencing with those richest in calcium salts.	
Spinach	...	Turnip-tops	. 37.15
Brussels sprouts	...	Sea-kale	. 27.56
Potato	...	Cauliflower	. 23.33
Asparagus	...	French beans	. 17.48
Savoy cabbage	...	Cabbage	. 17.14
French beans	...	Lettuce	. 15.02
Lettuce	...	Savoy cabbage	. 14.83
Beetroot	...	Turnip	. 13.38
Cabbage	...	Celery	. 13.06
Celery	...	Spinach	. 10.64
Turnip-tops	...	Carrot	. 6.88
Turnip	...	Brussels sprouts	. 6.16
Carrot	...	Potato	. 5.46
Cauliflower	...	Asparagus	. 5.05
Sea-kale	...	Green peas	. 4.98
Green peas	...	Beetroot	. 2.58

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium biurate bears no relationship, either of a direct or an inverse ratio, to the proportions of calcium salts present. For instance, it can be seen that the solvent effect on the biurate of the ash of potato is high, while the proportion

of calcium salts is low ; on the other hand, the solvent effect on the biurate of the ash of sea-kale is low, while the proportion of calcium salts is high.

Similarly it can be shown that the increased solvent effect on the biurate exerted by the mineral constituents of vegetables is not due to either the magnesium or iron salts present.

It can also be demonstrated that the increased solubility of the biurate is not due to the phosphates present in the vegetables. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of phosphates present.

TABLE XXI.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of phosphates present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of phosphates present, and showing the percentages of phosphates present in the ashes, reckoned as phosphoric anhydride, commencing with those richest in phosphates.	
Spinach	...	Green peas	. 35·62
Brussels sprouts	...	Cauliflower	. 22·14
Potato	...	Asparagus	. 21·93
Asparagus	...	Potato	. 15·99
Savoy cabbage	...	Carrot	. 15·02
French beans	...	Celery	. 14·39
Lettuce	...	Brussels sprouts	. 14·20
Beetroot	...	Savoy cabbage	. 13·19
Cabbage	...	French beans	. 12·21
Celery	...	Cabbage	. 11·99
Turnip-tops	...	Lettuce	. 9·62
Turnip	...	Turnip	. 9·26
Carrot	...	Spinach	. 8·56
Cauliflower	...	Beetroot	. 8·25
Sea-kale	...	Sea-kale	. 8·00
Green peas	...	Turnip-tops	. 6·15

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium bi-

urate bears no relationship, either of a direct or an inverse ratio, to the proportions of phosphates present. For instance, it can be seen that the solvent effect on the biurate of the ash of spinach is high, while the proportion of phosphates is low; on the other hand, the solvent effect on the biurate of the ash of green peas is low, while the proportion of phosphates is high.

It can also be demonstrated that the increased solubility of the biurate is not due to the sulphates present in the vegetables. The following table contains a comparison of the solvent powers exerted by the mineral constituents of vegetables on sodium biurate, and the proportions of sulphates present.

TABLE XXII.—*Showing that the solvent effect on sodium biurate of the mineral constituents of vegetables is not dependent on the amounts of sulphates present.*

Vegetables arranged in order of solvent effect of their mineral constituents on sodium biurate, commencing with those exerting the greatest effect.		Vegetables arranged in order of the proportions of sulphates present, and showing the percentages of sulphates present in the ashes, reckoned as sulphuric anhydride, commencing with those richest in sulphates.	
Spinach	...	Sea-kale	. 19·78
Brussels sprouts	...	Turnip-tops	. 15·27
Potato	...	Cauliflower	. 14·16
Asparagus	...	Savoy cabbage	. 12·85
Savoy cabbage	...	Turnip	. 12·47
French beans	...	Brussels sprouts	. 8·31
Lettuce	...	Cabbage	. 7·28
Beetroot	...	French beans	. 6·82
Cabbage	...	Potato	. 5·60
Celery	...	Asparagus	. 5·40
Turnip-tops	...	Carrot	. 5·20
Turnip	...	Spinach	. 4·44
Carrot	...	Green peas	. 4·36
Cauliflower	...	Lettuce	. 3·92
Sea-kale	...	Beetroot	. 2·41
Green peas	...	Celery	. 1·10

It is also evident from this table that the solvent effect of the mineral constituents of vegetables on sodium bi-





# ACUTE DILATATION OF THE HEART IN RHEUMATIC FEVER

BY

D. B. LEES, M.D.

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It is not, I think, sufficiently recognised that acute dilatation of the heart is a frequent, almost a constant, occurrence in a rheumatic attack. Attention has been too exclusively directed to the auscultatory phenomena as indicative of the presence of pericarditis or endocarditis, and it seems to have escaped notice that whether either or both of these inflammatory conditions be present, or whether both be absent, there is almost always in rheumatic fever more or less dilatation of the heart. As a part of the chronic cardiac disease caused by rheumatism dilatation is generally acknowledged to be of great importance, but it is too much regarded as merely one of the results of a valve-lesion or possibly of an adherent pericardium.

In studying the curative influence of the ice-bag in pericarditis I found it necessary to determine carefully day by day the limits of the præcordial dulness, and I was struck with the evidence of a rapid and often permanent dilatation of the heart accompanying the signs of pericarditis. In a paper on the "Treatment of Peri-

carditis," published in the 'Lancet,' July 22nd, 1893, I attributed this early dilatation to the weakening effect of the pericardial inflammation on the cardiac muscular wall.

But observation of the fact that cardiac dilatation is much less marked in pericarditis of renal origin, or of tubercular or suppurative nature, suggested a doubt whether this explanation of the dilatation accompanying rheumatic pericarditis was altogether adequate, and the subsequent discovery of cases of rheumatism in which marked dilatation of the heart was present without any evidence of either pericarditis or endocarditis proved that there must be some other influence at work.

In December, 1894, I had under my care in St. Mary's Hospital a young man of seventeen suffering from his first attack of rheumatism, in whom the præcordial dulness was greatly enlarged without any definite murmur and without any friction or other indication of pericarditis. I am aware that an audible rub is not invariably present in pericarditis, but the cases in which I have observed this have been of suppurative nature, and I doubt whether this complete absence of rub throughout the case ever occurs in rheumatic pericarditis. That the increase of the præcordial dulness was not due to effusion into the pericardium seemed to be proved by the palpable cardiac impulse, the distinctly audible heart-sounds, and the absence of dyspnœa and distress. Under treatment this increase in the dulness gradually became less, and returned to the normal. In 1896 I had three similar cases; in these I took tracings of the enlarged area of dulness, and again found that it diminished under treatment and gradually returned to the normal. One of these patients had been in the hospital under my care four months before (1895) with a previous attack of rheumatism, attended with great increase of the præcordial dulness without either rub or murmur, and on this occasion also the excess in the dulness had disappeared on his recovery.

Before showing these tracings as evidence of alteration in the outline of the heart one must decide whether per-

cussion is trustworthy as a means of determining with accuracy the size of the heart. If an opinion on this matter had to be formed from the statements in text-books, this would seem extremely doubtful; but the valuable papers of Dr. Ewart in the 'Lancet' of August 29th, 1891, and the 'British Medical Journal' of March 21st, 1896, show that the true outline of the heart can be much more precisely ascertained than most physicians have supposed, while the paper read by Dr. Herringham before the British Medical Association at Carlisle in July, 1896, proves that the results thus obtained are almost exactly verified by post-mortem examination.

Confusion is introduced into this question by the terms "superficial" and "deep." What is called "superficial cardiac dulness" is of no *cardiac* value whatever. It is of importance as indicating the condition of the left lung, whether on the one hand it is emphysematous or on the other shrunk and fibrosed. But it would be much better to speak of the "uncovered cardiac area." The term "cardiac dulness" would then be understood to indicate the real size of the heart, a fact of the greatest importance.

In this way we should get rid also of the term "deep," which is apt to suggest that the percussion required to determine the true cardiac outline must be of a forcible kind. This is the very reverse of the truth, for such percussion brings out the pulmonary and gastric resonances and defeats its own object. Light percussion is absolutely necessary, especially over the sternum, which very readily conducts resonance from the lungs. If this be constantly kept in mind, there is usually little difficulty in defining with very considerable accuracy the outline of the heart on both left and right sides, for the cardiac margin on both sides is comparatively thick, and the alteration of the note on light percussion at the margin is generally quite definite.

It is well to begin in the region of the apex, and to remember that the left limit of the cardiac dulness extends

beyond, in dilatation often much beyond, the position of the impulse. The precise position of this limit should be found in the fourth and in the fifth spaces, sometimes in the sixth also.

Next, the extension towards the right of the right auricle should be determined by very careful light percussion to the right of the sternum in the fourth space, just below the line joining nipple to nipple. In the fifth space it is somewhat more difficult, because the partial hepatic dulness is here present also, but it can usually be defined satisfactorily. In health there is always about one fingerbreadth of dulness to the right of the sternum in the fourth space, in accordance with the anatomical facts.<sup>1</sup> When the auricle is dilated, as in mitral stenosis, the limit may be two or even three fingerbreadths to the right of the sternum. This is a most valuable indication of the condition of the right auricle, and one too much neglected; it is often of great assistance in determining whether removal of blood, by venesection or by leeches, is desirable in mitral disease or in pneumonia.

I measure by fingerbreadths rather than by inches or centimetres, for the use of these measures would imply that the limit can be determined with mathematical precision, which is not the case; no more than a close approximation can be claimed. The measure by fingerbreadths is made during the routine examination by percussion, it is made without any marking of the patient's skin, and without exciting his attention; it is a measure which the physician always carries with him, and it is invariable for the same observer.

Having determined the extreme cardiac limit both to left and to right, the position of the lateral margins of the heart may easily be defined if it be remembered that they both slope upwards and inwards, and the percussed finger be held in a position parallel to this slope in each case. The right margin above the nipple-level rapidly

<sup>1</sup> See also the "radiographs" of the cardiac outline by Dr. Williams of Boston, '*Brit. Med. Journ.*,' 1898, vol. i, 1006.

approaches the sternum. The left margin normally rises to the inner side of the nipple, but in a moderately dilated heart the limit of dulness will be found to pass through the nipple, and where the dilatation is great it may cross the vertical nipple-line at one, two, in extreme cases even three fingerbreadths above the nipple. A little practice gives facility in determining the opposite borders of the heart, so that even without any marking of the skin one seems almost to *see* the outline of the heart. But a permanent record can easily be obtained by marking the outline with a blue pencil and taking a copy of this on tracing-paper, care being taken to indicate in all cases the median line, the infra-costal angle, and the position of the nipple, as points of reference. The extension of the cardiac dulness to left and to right of the median line may then be measured in inches or centimetres.

The upper and lower limits of the cardiac outline are less easy to determine satisfactorily. Dr. Ewart's observations about them, in the papers already referred to, will repay perusal. I have usually refrained from attempting to include the lower limit in the tracings, and I lay no stress on the accuracy or the precise meaning of the upper limit of the dulness, for the sternum and the great vessels here cause difficulty.

It may be well to add that the precise determination of the limit of the heart to right or to left may be impossible when there is fluid in the pleura, and it may be difficult when the breasts are large, when there is consolidation of parts of the lungs adjacent to the heart, or when emphysema exists. Fortunately, emphysema is rarely present in the subjects of acute rheumatism, who are usually in the earlier half of life. But if emphysema, pleurisy, and consolidation of lung are absent the lateral outline of the heart obtained by careful light percussion is almost absolutely correct, as I have found by post-mortem investigation. If the outline determined by percussion be marked on the cadaver, and long needles be passed through various points in the bounding line on either

side, it is found that they correspond with surprising accuracy to the margin of the heart.

It seems to me very important that a determination of its exact size by careful light percussion should invariably form a part of the routine examination of the heart. For it is dilatation that is the enemy, and an exact knowledge of the size and strength of the heart is far more important than the most elaborate and minute study of murmurs. It is very necessary in typhoid fever, where the cardiac condition is of great importance; it is indispensable in influenza and in diphtheritic paralysis, in both of which sudden death may occur from syncope; it may save life by indicating the need for bloodletting when the right heart is becoming paralysed from over-distension in pneumonia, capillary bronchitis, or mitral disease. And surely in rheumatic fever, a disease known specially to injure the heart, it ought never to be neglected. Yet are we not all too apt to use our stethoscope and neglect percussion, forgetting Sir George Humphry's rule, "*Eyes first, fingers next, ears last*"? We are prone to be satisfied if no murmur is audible. Yet in the absence of murmur there may be great dilatation; in proof of this I submit the following tracings.

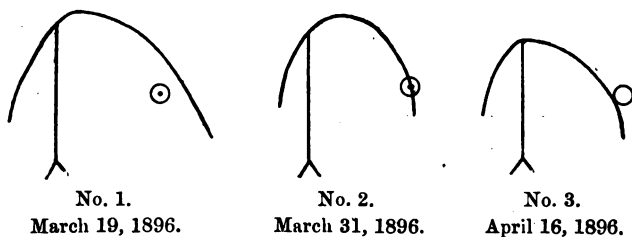
The first three sets of tracings are taken from the cases of three young men suffering from their first or second attack of rheumatism, in none of whom any rub or definite murmur was audible, but in all of whom the cardiac dulness was for a time considerably enlarged. In all three the cardiac dulness extended one and a half or two fingerbreadths to the left of the nipple line, and one and a half or two fingerbreadths to the right of the sternum, on their admission into hospital; in all three it shrank to the normal dimensions as the rheumatic symptoms disappeared under treatment. I now show three tracings in each case, the first taken on commencing treatment, the third on recovery, and the second at an intermediate stage. Other tracings were taken, and these manifest a gradual reduction, but for the sake of

clearness I show only three in each case. *Every tracing was taken without reference to previous diagrams of the same case*, so as to eliminate the element of bias as much as possible. They have been carefully reduced in size on the same scale by Dr. Poynton, in order to make it possible to throw them on a screen by a lantern. The first tracing in each case has been coloured black, the second red, the third blue, so that the reduction in size is at once obvious.

Of course in all diagrams of this kind allowance must be made for some unavoidable exaggeration due to the fact that the tracing is taken on the curved surface of the thorax and is then flattened out on a plane surface. So that the transverse diameter of the diagram is necessarily somewhat longer than the actual transverse diameter of the heart.

CASE 1.—G. G—, aged 34, admitted into St. Mary's Hospital March 19th, 1896, suffering from slight arthritis of the left wrist and both shoulders. Temperature, on admission  $100\cdot5^{\circ}$ ; on the next day  $99^{\circ}$ ; on the third day  $98^{\circ}$ . Treatment, sodium salicylate 20 grains every three hours.

G. G—, aged 34. Rheumatic fever.



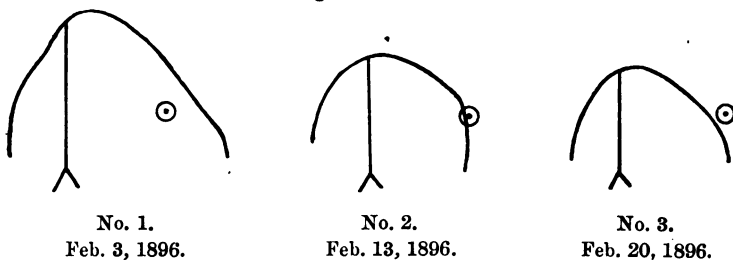
The first tracing shows the area of cardiac dulness on his admission.

The second tracing was taken twelve days after the first; the left border of the dulness has now retreated to the nipple-line.

The third tracing was taken sixteen days after the second, a month after the first; the left border is now well within the nipple-line. The right border shows also a return to the normal.

CASE 2.—Alfred H—, aged 16 years, admitted February 3rd, 1896. Slight affection of ankles, knees, and hip-joints. Temperature normal. After four 20-grain doses of salicylate, the treatment was by 40-grain doses of sodium carbonate every two hours; this was continued for a fortnight.

Alfred H—, aged 16. Rheumatic fever.



The first tracing shows the cardiac area on admission.

The second tracing was taken ten days after the first; it shows a diminution of two fingerbreadths in the left margin of the heart, the border now passing through the nipple.

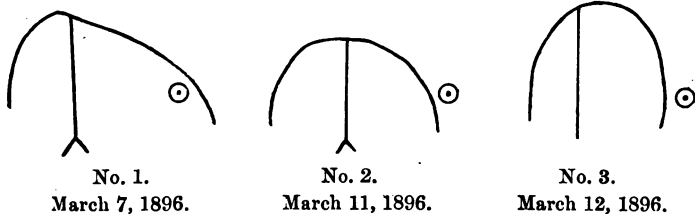
The third tracing was taken seven days after the second; the heart has now nearly returned to its normal dimensions.

CASE 3.—Albert R—, aged 21 years, admitted March 6th, 1896. Slight arthritis of left knee, ankle, and shoulder. Temperature  $103^{\circ}$  on first evening,  $99^{\circ}$  next morning; some irregular pyrexia continued for fifteen days. After about six 20-grain doses of salicylate the treatment was by 40-grain doses of sodium carbonate hourly during the daytime.



The first tracing shows the cardiac area on admission.  
The second tracing was taken four days after the first,

Albert R—, aged 21. Rheumatic fever.



and shows a marked shifting inwards of the left edge of dulness; but the area of dulness towards the right has somewhat increased. This was apparently due to an attack of pleurisy, pleural friction being plainly heard. Leeches were applied, and in the third tracing, taken on the following day, the right border of the heart as well as the left has returned to the normal position. I have already said that I lay no stress on the upper limit of these tracings, and I think it possible that the upper limit of this third tracing may be erroneous.

This patient had been under my care four months before, during a previous attack of rheumatism. On this occasion also he had acute dilatation without pericarditis or endocarditis. At first the cardiac dulness extended to one and a half fingerbreadths, or one inch, outside the nipple line. On his recovery it had become normal, and was found half an inch internal to the nipple.

The fourth set of tracings shows the supervention of acute dilatation in a heart already damaged by previous rheumatism.

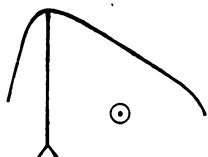
CASE 4.—Harriet W—, aged 14 years, admitted into St. Mary's Hospital, March 10th, 1896, suffering from her third attack of rheumatism. The first attack occurred four years before, the second three years before; in both chorea accompanied the rheumatic symptoms. This third

attack commenced three weeks ago; in it there had been dyspnœa, pain referred to the heart, also pain in the elbows and feet.

On admission temperature  $99^{\circ}$ , pulse 122, respirations 24. Fluid in both knees, ankles also swollen. Faint systolic murmur at apex, and over pulmonary artery; suspicion of pre-systolic murmur at apex. Patient emotional, but not distinctly choreic. Urine faintly acid. No salicylate was employed in this case, the treatment being by 30-grain doses of sodium carbonate given hourly during the daytime.

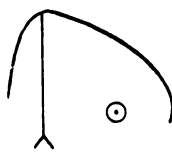
The first tracing was taken the day after her admission; it shows the left border of the heart extending three fingerbreadths outside the nipple-line, and two and a half above the nipple, while the right border is fairly normal.

Harriet W—, aged 14. Rheumatic fever.



No. 1.

March 11, 1896.



No. 2.

March 12, 1896.



No. 3.

March 16, 1896.

The second tracing, taken the next day, is identical with the first on the right side, but shows a marked reduction on the left side. The pre-systolic murmur was now distinct.

The third tracing, taken four days after the second, is similar to the others on the right side, but shows a further large reduction on the left side. The urine was now strongly alkaline. A fourth tracing, taken nine days after the third, is practically identical with it; she was now taking the alkaline medicine every three hours, and the urine had become again faintly acid.

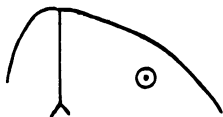
The third and fourth tracings represent doubtless the

permanent increase in size of her heart caused by the previous disease, which had left behind it distinct slight stenosis and regurgitation at the mitral. The first tracing shows the additional temporary acute dilatation of the present attack of rheumatism.

The fifth set of tracings shows the effect of the combination of acute dilatation with acute pericarditis.

CASE 5.—Daisy D—, aged 9 years, was admitted into St. Mary's Hospital February 7th, 1896, suffering from chorea, pericarditis, and slight arthritis of hands and feet. This was her second rheumatic attack. Twelve months previously she had been an in-patient for rheumatism, followed by chorea. On her admission a loud pericardial rub and a systolic apex murmur were heard by the house physician, who at once applied an ice-bag over the heart, and ordered 10-grain doses of sodium salicylate with fifteen grains of sodium carbonate every four hours. I saw her the next day and took the first diagram, which

Daisy D—, aged 9. Rheumatic fever.



No. 1.

Feb. 8, 1896.



No. 2.

Feb. 21, 1896.

shows an enormous increase of the transverse cardiac dullness, the left border extending to more than three finger-breadths outside the nipple-line, and about two above the nipple; the right border also is much too far to the right. It is not unlikely that part of this increased dullness was due to the presence of some pericardial effusion. The medicine was repeated every two hours. Next day the frequency was altered to every three hours, and on the following day (10th) to every four hours. On the

11th the urine was found to be still acid, and again on the 13th it continued acid, so that the medicine was again given every three hours. On the 14th the pericardial rub had disappeared and there was no longer any chorea, but a loud systolic murmur was heard at the apex, and pleuritic friction was detected in the right axilla. The medicine was therefore given every two hours, and a second ice-bag was placed over the anterior base of the right lung. Two days later, the urine being still acid, the dose of sodium carbonate was doubled, and as the pleurisy had disappeared the second ice-bag was removed from the right lung. On the 19th the urine had at last become alkaline. On the 22nd the ice-bag over the heart was removed, having been applied continually for fifteen days.

The second tracing was taken on the 21st, thirteen days after the first. It shows an extraordinary diminution in the cardiac dulness, and the improvement in the general condition corresponded to the improvement in the tracing. The pericarditis had subsided, but evidence of endocarditis remained. The second tracing probably indicates the permanent cardiac enlargement produced by the first rheumatic attack. The great increase in the præcordial dulness shown in the first tracing may have been due in part to effusion into the pericardium, but was probably mainly due to acute dilatation.

It is worth while to point out incidentally that in this case, in spite of the pericarditis and the dilatation, the free and prolonged administration of salicylate of sodium produced no "depressing" effect.

I do not claim that these tracings are absolutely accurate; no doubt they are open to criticism in points of detail, and I would again refer to Dr. Ewart's valuable papers. But they were taken with care, in all cases by myself, usually verified by another observer, *and always without reference to the preceding tracing.*

I think they are sufficient to prove that an acute dilata-

tion of the heart does occur in acute rheumatism; that it may be marked even when there is only slight pyrexia and but little arthritis, with no modification of the cardiac auscultatory phenomena beyond slight alteration in character of the heart-sounds, and that it may complicate the evidence of former heart-disease or of fresh endocarditis or pericarditis.

I made a preliminary statement of these facts in a letter to the 'Lancet' of July 25th, 1896, and also mentioned therein that a similar dilatation occurs in most cases of chorea, even when no murmur exists,—a new evidence of the essentially rheumatic nature of chorea. Since that date I have made other observations on rheumatism in adults at St. Mary's Hospital, and have obtained results similar to those already exhibited. I have made observations also on the occurrence of acute dilatation of the heart in the rheumatism and chorea of childhood at the Hospital for Sick Children, mainly in conjunction with my friend Dr. Poynton, formerly house-physician at that hospital, and now medical registrar and pathologist at St. Mary's Hospital. We have embodied our results in a joint paper, illustrated with tracings taken very carefully by Dr. Poynton and confirmed in my own cases by myself. It seemed desirable that the results should be compared with observations on the hearts of children free from rheumatism and cardiac disease. Dr. Poynton therefore took tracings of the cardiac outline in forty-five children in the surgical wards at Great Ormond Street during his subsequent tenure of office as house-surgeon. He has also carefully examined the hearts of thirty-five healthy boys, aged twelve and thirteen, at Marlborough School, with the kind permission of Dr. Penny. Finally, he has analysed the post-mortem records of 150 cases of fatal rheumatic heart-disease in children under twelve.

I venture to think that the pathological and clinical results of his researches and observations afford additional and decisive proof of the frequency and the serious importance of rheumatic dilatation of the heart.

The occurrence of acute dilatation of the heart in rheumatism has not been entirely overlooked. Thus Dr. Sansom writes ('International Clinics,' 1894, p. 7): "I am convinced, however, that the rapid increases of dullness over the heart in rheumatism are not all due to pericardial inflammation and the effusion of fluid; the whole heart may become swollen and dilated—swollen with the products of inflammatory exudation, dilated because of the enfeeblement of the muscle of its right and left chambers. In some cases this condition of swollen heart disappears without any of the friction signs of pericarditis being manifested; in fact, the heart and its serous membranes may pass through changes like those occurring in a joint inflamed through rheumatism. These variations in the bulk of the heart may be observed in some cases to be considerable from day to day, and there may be repeated enlargement at intervals of a few days, just as there may be repeated swelling in the joints."

Dr. Samuel West reported to the Pathological Society in 1882 the case of an anæmic boy of sixteen, who died two months after an attack of rheumatic fever, and was found to have dilatation of all the cardiac cavities, especially of the ventricles. Evidence of endocarditis also existed. The myocardium was affected with fatty degeneration.

At a still earlier date, in 1879, Dr. Goodhart exhibited to the same society the heart of a boy of seventeen who died within a month after an attack which was certainly rheumatic, in whom the heart had rapidly enlarged. The left ventricle was "rather widely dilated." Weight of heart, 19 ounces; pericardium entirely adherent by soft lymph; recent valvulitis; muscle slightly fatty.

In the same year, in the 'Guy's Hospital Reports,' Dr. Goodhart described cases of acute dilatation of the heart following scarlet fever, and another was published in 1880 by Dr. Barlow in the 'Medical Times and Gazette' (1880, vol. i, p. 426). In none of these, however, was

there any evidence of rheumatism. But in a note appended to his paper Dr. Goodhart adds: "Sir William Gull was in the habit of giving special prominence to the fact that acute pericarditis in rheumatic fever is liable to give rise to rapid dilatation of the heart, which is often mistaken for pericardial effusion. Sir W. Gull also taught that dilatation is succeeded, not preceded, by hypertrophy in some of these cases." "I am very glad," Dr. Goodhart continues, "to be able to mention this, not only because it is a weighty confirmation of some of the remarks made in this paper from another point of view, but also because it has hitherto been unrecorded."

It may perhaps be doubted whether the value of these observations of Sir William Gull's has been adequately appreciated. At any rate, I think that the frequency and extent of acute cardiac dilatation in rheumatic fever is insufficiently recognised by most physicians. Yet it is a condition of great gravity, and its presence or absence ought to be carefully ascertained in every case of the disease. When uncomplicated with pericarditis or endocarditis, if the patient be kept absolutely at rest it may prove transitory, and the heart may entirely recover itself, especially under appropriate medicinal treatment. But when it complicates pericarditis, it adds enormously to the danger of the latter condition. In the more severe cases of rheumatic pericarditis, the accompanying acute dilatation is probably largely responsible for the dangerous symptoms of cardiac failure, for the dyspnoea, the tendency to cyanosis, the feeble pulse, and the delirium.

When the pericarditis is over, and has ended in pericardial adhesion, the heart becomes fixed in its dilated state, and it is never again able to return to the normal. Some amount of hypertrophy may follow, enough to maintain compensation, in the absence of much exertion, for a limited time; but such hearts are permanently crippled and soon break down. Cases of this kind are only too frequent in children and young adults.

Acute dilatation is probably a much more important

factor than endocarditis in the production of many cases of chronic heart disease. In a girl recently under my care at Great Ormond Street, enormous dilatation of the heart was produced in six months after the first illness. She died from pericarditis ; it was found after death that there was no fluid in the pericardial sac, but recent adhesions everywhere. There was some mitral endocarditis, but it was very slight, and entirely inadequate to account for the remarkable dilatation of the ventricles. If this patient had survived, and come under observation at a later period with an enlarged heart and a systolic apex murmur, it is almost certain that the cardiac dilatation would have been considered the consequence of her mitral insufficiency. It is probable that very many cases of chronic heart-disease now attributed to "mitral regurgitation" or to "adherent pericardium" are essentially the permanent results of an acute dilatation, the pericardial adhesions and the valvular damage taking but a small share in the morbid process.

It is unlikely that many cases of the acute dilatation above described are due to definite myocarditis. No doubt this is a possible cause, and it was present in a case recently reported (with autopsy) to the Clinical Society by Dr. Herringham. But the transitory nature of the affection under appropriate treatment, as shown by the tracings now exhibited, seems to prove that in these there cannot have been any actual inflammation, though there may have been an acute congestion, of the muscle. It is possible that skilled microscopical examination may reveal some change in the nutritive condition of the cardiac muscular fibre generally, but whether this be so or not, it seems likely that the effect is a toxic one.

Sixteen years ago ('Journal of Physiology,' vol. iii) Dr. Gaskell proved that a dilute solution of sodium hydrate caused gradual progressive contraction of the frog's ventricle until it remained persistently fully contracted and failed to relax at all ; it caused also a similar contraction of the arterioles. On the other hand, a dilute



solution of lactic acid caused relaxation and "extreme dilatation" of the ventricle, and finally diastolic standstill; it caused relaxation also of the vessels. He investigated the action of various drugs, and found that some of them acted like soda, others like lactic acid.

These experiments seem to suggest that the production of the remarkable cardiac dilatation which occurs in rheumatism may be due to the presence in the circulating blood of a poison acting on the heart like lactic acid. May not this be the toxine resulting from the development of a micro-organism? It can hardly be doubted that this is the true explanation of the acute and sometimes fatal dilatation of the heart which occurs in influenza. The more carefully rheumatism in childhood is studied, the more reason will be found for the belief that it cannot be due to any mere perversion of metabolism, but must be due to some microbic process.

The suggestive observation of Dr. Gaskell as to the influence of soda in causing cardiac contraction seemed to me to afford reason for hope that this alkali might be found of service in diminishing the dilating effect of the rheumatic toxine. I have therefore treated some cases with large and frequent doses of sodium carbonate, and I have thought that a more rapid shrinking of the enlarged area of cardiac dulness occurred in these cases than in those treated with salicylates alone. The plan seems to me worthy of further trial. At all events I found that the drug was well tolerated in spite of its unpleasant taste, that it never caused vomiting or lessened appetite in rheumatic subjects, and that large doses were required to render the urine alkaline. I selected the carbonate in order to secure as large an amount as possible of the sodium element, forgetting for the time that the water of crystallisation contained in it makes it really less powerful as an antacid than the bicarbonate. I am now making further observations upon the action of the bicarbonate.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 189.



ACUTE DILATATION OF THE HEART  
IN THE  
RHEUMATISM AND CHOREA OF  
CHILDHOOD

BY

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Received April 5th—Read June 28th, 1898

THIS paper embodies the results of our joint investigation of the subject of acute dilatation of the heart in rheumatism and chorea made at the Hospital for Sick Children, and it also includes an account of certain inquiries conducted by Dr. Poynton in further elucidation of the subject. These inquiries comprise—

1. Observations on the size, strength, and sounds of the heart in healthy children.

2. Observations on the condition of the heart in rheumatic and choreic children under the care of physicians other than Dr. Lees. We tender our thanks to Dr. Cheadle, Dr. Barlow, Dr. Phillips, and Dr. Penrose, for kind permission to make use of their cases.

3. An analysis of 150 fatal cases of rheumatic heart disease in children under twelve years of age, taken from the records of the Hospital for Sick Children and from those of St. Mary's Hospital.

Before proceeding to discuss the condition of the heart in rheumatic and choreic children we wish, in order to obtain a standard of comparison, to draw attention to observations made by Dr. Poynton on the size, strength, and sounds of the heart in children free from rheumatism. These were carried out partly in the surgical wards of the Hospital for Sick Children, in forty-five cases under the care of Mr. Owen, Mr. Morgan, and Mr. Pitts, who kindly allowed us to use their patients; and partly at Marlborough College, where the hearts of thirty-five healthy boys of twelve and thirteen years were examined by the permission and with the kind assistance of Dr. Penny, medical officer to the College, to whom also we offer our thanks.

All these observations were made with the child in the same posture—on the back, with the left arm abducted. The results are given in detail in Appendices A and B. We may briefly summarise them as follows:

In children aged from seven to twelve years the area of cardiac dulness (by which we mean the nearest approach to the actual size of the heart that can be obtained by percussion) extends upward to the third costal cartilage on the left side, thence downward and to the left to the fourth space just internal to the nipple, or even as far as the nipple-line. Crossing the middle line above, it extends in the fourth right space three quarters of an inch to the right of the median line, a bare fingerbreadth to the right of the sternal margin. As it passes downward it tends slightly inward before it reaches the hepatic dulness, and then recrosses the middle line to reach the apex.

In children under seven years of age the cardiac dulness extends as far as the left nipple-line more frequently than is the case in older children.

In the boys between twelve and fourteen, the left limit

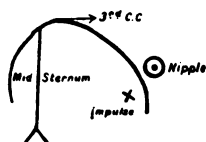
of the cardiac dulness was almost invariably distinctly internal to the left nipple-line, on the average about three quarters of an inch from it, and reached the fifth left rib or more frequently the fifth space.

In healthy children the cardiac impulse is usually internal to the left limit of the dulness, often markedly so. The action of the heart is regular. At the apex, the first sound is longer than the second. At the base, the second sound is louder on the left side than on the right, though both have a distinct relative sharpness. Soft blowing systolic murmurs are sometimes heard, most frequently in the fourth space internal to the impulse, sometimes at the base, more rarely at the apex; they may be modified by position. When these murmurs occur, the area of the heart is often rather larger than normal, the general physique more feeble, and the child anæmic.

So far as regards the position of the cardiac impulse and its relation to the nipple, in younger and in older children, our results are in accord with those of Stärck and of Dr. Archibald Garrod quoted by the late Dr. Sturges in the Lumleian lectures in 1894. But in determining the "cardiac dulness" we have rejected the "superficial dulness" and endeavoured to ascertain the true outline of the heart.

We conclude this section of our paper by showing with the lantern the area of cardiac dulness in a healthy boy

Healthy boy, aged 12.



of twelve, to serve as a standard of comparison for the tracings taken from rheumatic and choreic children.

We now proceed to show tracings taken from the hearts of children suffering from rheumatism or from chorea,

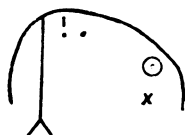
and would premise that *the several tracings in each case were always taken without reference to previous tracings from the same case.*

The varying severity of the cardiac affection makes it desirable to arrange our observations in four groups.

GROUP I.—*First attacks of mild subacute rheumatism, in which there was no pericarditis, and either no murmur or only a systolic murmur best heard internal to the nipple.*

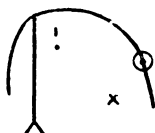
CASE 1.—E. S—, aged 12, admitted into St. Mary's Hospital under Dr. Cheadle October 9th, 1897, for moderate articular rheumatism.

E. S—, aged 12. Rheumatic fever, first attack.



No. 1.

Oct. 10, 1897.



No. 2.

Oct. 26, 1897.

! means accentuated pulmonary second sound. . means systolic murmur.  
x means impulse.

October 10th.—First tracing. Temperature normal. Impulse in the V.N.L. Area as shown. Short first sound. Accentuated pulmonary second.

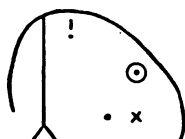
16th.—A slight rise of temperature (to 99·8°), and a soft systolic murmur heard internal to the impulse.

26th.—Second tracing. Steady recovery had taken place. Temperature normal. Impulse internal to V.N.L. Area as shown. Systolic murmur very faint. It disappeared two days afterwards.

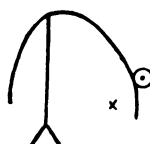
CASE 2.—F. D—, aged 11, admitted into St. Mary's under Dr. Lees, April 6th, 1897, with mild articular rheumatism.

April 7th.—First tracing. Temperature  $102^{\circ}$ . Impulse diffuse. Area as shown. Soft systolic murmur internal to impulse. Loud pulmonary second.

F. D—, aged 11. Rheumatic fever, first attack.



No. 1.  
April 7, 1897.



No. 2.  
April 17, 1897.

! means accentuated pulmonary second sound. • means systolic murmur.  
x means impulse.

8th.—Temperature fell to normal.

17th.—The boy well. Second tracing. Impulse more definite, and internal to the nipple. Area diminished. No murmur.

CASE 3.—E. D—, aged 9, admitted into the Hospital for Sick Children under Dr. Lees, February 29th, 1896, for articular rheumatism.

E. D—, aged 9. Rheumatic fever, first attack.



No. 1.  
March 1, 1896.



No. 2.  
March 5, 1896.



No. 3.  
April 7, 1896.

! means accentuated pulmonary second sound. • means systolic murmur.  
x x means diffuse impulse.

March 1st.—First tracing. Temperature  $101.2^{\circ}$ . Impulse diffused, and external to V.N.L. Faint systolic murmur internal to the impulse. Loud pulmonary second.

5th.—Second tracing. Increase in cardiac area. Salicylates were now pushed, and the dilatation slowly subsided.

April 7th.—Third tracing. Impulse definite and internal to V.N.L. Area diminished. Systolic murmur gone.

GROUP II.—*First attacks of chorea without history of previous rheumatism.*

The occasional occurrence of dilatation of the heart in chorea has been noted both by Dr. Garrod and by Dr. Osler. We find that it is common, even in cases in which there is no indication of rheumatism other than the chorea and no history of any previous rheumatic attack. In thirty-three cases without history of previous rheumatism, and in many of them without evidence of present rheumatism, we found that the area of cardiac dulness extended to the left of the nipple-line (usually about one finger-breadth) in no fewer than twenty-nine, and in sixteen of these the impulse also was external to the nipple. On the other hand, in only three of them was there evidence of increase of the cardiac dulness towards the right. The auscultatory signs were noted in twenty-eight cases; in twenty-four of these the first sound was short, or accompanied by a faint systolic murmur. Tracings were taken in all the thirty-three cases, but a day was always allowed to elapse after the child's admission into hospital before the first determination of the cardiac outline was made. We now show two examples illustrating the condition of the heart in typical cases.

CASE 4.—W. A—, aged 12, admitted into St. Mary's Hospital, October 19th, 1897, under Dr. Lees, for chorea of moderate severity.

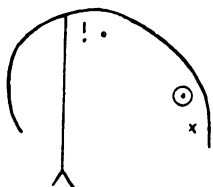
October 20th.—First tracing. Temperature 100·2°. Heart's action irregular. Impulse external to nipple. Area as shown. First sound remarkably short. Systolic murmur over pulmonary artery; loud pulmonary second.



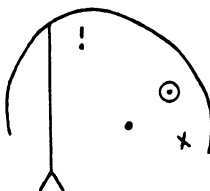
During the early part of November there was some irregular pyrexia, and a rheumatic erythema appeared.

November 19th.—Second tracing. The area had increased, and a soft blowing murmur could be heard internal to the nipple. After this there was gradual and slow recovery.

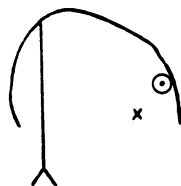
W. A—, aged 12. Chorea, first attack.



No. 1.  
Oct. 20, 1897.



No. 2.  
Nov. 19, 1897.



No. 3.  
Jan. 3, 1898.

! means accentuated pulmonary second sound. . means systolic murmur.  
x means impulse.

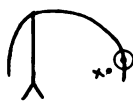
January 3rd, 1898.—Third tracing. Area diminished. Chorea and murmur gone.

CASE 5.—M. M—, aged 10, admitted into the Hospital for Sick Children, March 17th, 1896, under Dr. Penrose. Chorea distinct, and of five weeks' duration.

M. M—, aged 10. Chorea, first attack.



No. 1.  
March 18, 1896.



No. 2.  
April 4, 1896.



No. 3.  
April 9, 1896.

x x means diffuse impulse. x means impulse. . means systolic murmur.

March 18th.—First tracing. Temperature 99°. Impulse just internal to V.N.L. Area as shown. Basic systolic pulmonary murmur; accentuated second sound.

April 4th.—Second tracing. Chorea nearly well. Area of dulness much diminished.

9th.—Third tracing. The child had been getting up, but on the 8th had a little pyrexia, and a relapse of chorea. She was sent back to bed. This tracing shows an increase in the area.

She subsequently entirely recovered.

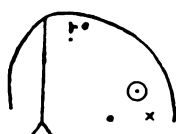
It is clear, therefore, that in first attacks of rheumatism and in first attacks of chorea there is often a definite increase in the cardiac area, appearing and disappearing under observation; there is also an outward movement of the impulse, and an accentuation of the pulmonary second sound; sometimes there is development of a soft systolic murmur internal to the nipple; occasionally there is irregularity. Evidence of active rheumatism may be present also; such as arthritis, or erythema, or there may be pyrexia alone. It is in such a combination of signs, more or less developed, that such hearts differ from the normal standard; it is a difference which is distinct, and the earliest appreciable in the history of rheumatic heart-disease. It cannot be explained by pyrexia, for it may be present when the temperature is normal; nor by the effect of salicylates, for it is present before treatment has been commenced; nor in cases of chorea by the movements, for its amount bears no constant relation to the severity of these. It is not merely a part of the debility caused by an illness, for it is often much more distinct in very mild attacks of rheumatism than in more severe diseases. It is evidently in some way a special result of the rheumatic process. The evidence already given appears to prove that it is independent of pericarditis. It is more difficult to prove the absence of endocarditis, but if any valvulitis at all was present in the above cases it must have been extremely slight, and quite incapable of producing so definite an enlargement of the heart, or one capable of such easy recovery. It seems impossible to avoid the conclusion that in rheumatism there is some

toxic action exerted on the cardiac muscle, enfeebling it and causing it to give way before the normal blood-pressure. This explains why the first sound becomes short, the area of dulness increased, and the impulse diffused. The feebler diastolic rebound, causing a weaker suction action in diastole, explains why the pulmonary tension rises, and the pulmonary second becomes accentuated.

Before passing to the more severe rheumatic cases, which we have placed in the third group, we wish to give an example of acute dilatation in a first attack of chorea, which was followed by definite valvulitis.

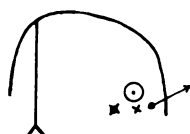
CASE 6.—L. H—, aged 11, admitted into hospital August 19th, 1897, under Dr. Lees, for moderate chorea of two weeks' standing.

L. H—, aged 11. Chorea, first attack.



No. 1.

Aug. 20, 1897.



No. 2.

Aug. 30, 1897.

! means accentuated pulmonary second sound. . means systolic murmur.  
 → means systolic murmur conducted to axilla. x x means diffuse impulse.

August 20th.—First tracing. Temperature  $99^{\circ}$ . Impulse external to nipple. Area as shown. First sound short. Sounds spaced. A very faint soft murmur heard internal to the impulse.

This condition continued unchanged for ten days. On the 29th the temperature rose from normal to  $99.8^{\circ}$ , and a definite musical blowing murmur appeared.

30th.—Second tracing. Area as shown. The murmur could be traced to the axilla, and all who examined the case agreed that there was now definite valvulitis.

When the patient left the hospital a month later, the murmur was still present and audible in the axilla.

GROUP III.—*Severe cases of acute rheumatic heart disease, with definite valvulitis and frequently pericarditis.*

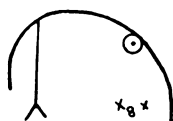
We give tracings from two cases as types of these severe attacks.

CASE 7.—E. C—, aged 11, admitted into the Hospital for Sick Children, February 20th, 1896, under Dr. Penrose, for a severe first attack of chorea. In the out-patient department in the morning no murmur was heard, but later in the day a soft apical murmur appeared.

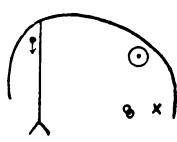
E. C—, aged 11. Chorea and rheumatic fever, first attack.



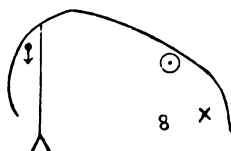
No. 1.  
Feb. 21, 1896.



No. 2.  
Feb. 29, 1896.



No. 3.  
March 10, 1896.



No. 4.  
April 22, 1896.

↓ means diastolic murmur. 8 means to-and-fro mitral murmur.  
x means impulse.

February 21st.—First tracing. Temperature 101°. Impulse external to nipple. Area as shown. A to-and-fro murmur audible external to the nipple. Loud pulmonary second.

23rd.—Erythema.

27th.—Arthritis.

29th.—Second tracing. Temperature  $99^{\circ}$ . Area slightly diminished. For some days the aortic second had been short.

March 10th.—Third tracing. Area no larger than before, though a well-marked aortic diastolic murmur had developed. The rheumatism had quieted down.

April 22nd.—Fourth tracing. After decided improvement there were now fresh pyrexia and joint pains. Area decidedly increased. Signs of aortic disease less marked.

Eventually the boy recovered sufficiently to leave the hospital, but with marked aortic and mitral regurgitation.

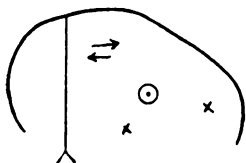
In this case there was no evidence of pericarditis. The increase in the cardiac dulness was most marked when the rheumatism was most active, and it diminished when the rheumatism subsided. With the fresh outburst the area again became enlarged. If the dilatation had been due to the valvular lesions, it would not have varied in this way, but would have steadily and gradually increased until checked by compensatory hypertrophy.

CASE 8.—E. B—, aged 8, admitted under Dr. Penrose April 11th, 1896, for general pericarditis and chorea.

April 12th.—First tracing. Temperature  $99^{\circ}$ . No definite impulse; area as shown; loud general friction. Very ill.

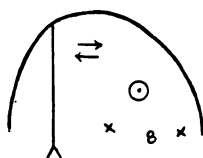
17th.—Sharp râles over front of left lung.

E. B—, aged 8. Chorea and rheumatic fever, first attack.



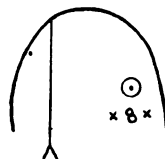
No. 1.

April 12, 1896.



No. 2.

April 18, 1896.



No. 3.

April 29, 1896.

± means pericardial friction. x x means diffuse impulse.

8 means to-and-fro mitral murmur.

18th.—Second tracing. Area diminished ; less friction ; a double murmur at the apex.

29th.—Third tracing. Remarkable improvement ; chorea almost gone. Area still further diminished. No friction ; double murmur plainly heard.

June 8th.—Discharged ; only a loud systolic murmur remains ; area still further diminished.

In this case, when the area of cardiac dulness was most extensive, the pericardial friction was loudest and most general. This suggests that there was probably no great excess of fluid in the pericardial cavity. The suggestion is supported by the post-mortem records of fatal cases of rheumatic heart disease. In only 12 out of 150 cases (see Appendix C) is it definitely stated that more than two ounces of fluid were found in the pericardial cavity. By actual experiment on the cadaver we have ascertained that this quantity will not produce anything like the enlargement of the cardiac area often observed during life. Dr. Sibson found that six ounces of fluid were required to distend the pericardium in a boy of nine. If to these facts we add the clinical observation that the area of pulsation, in these cases, is usually extensive and the cardiac sounds fairly loud, we are driven to the conclusion that in the great majority of cases of rheumatic pericarditis the increase in the area of cardiac dulness is mainly due to dilatation. At first sight it might seem that the dilatation is secondary to the pericarditis, but it is much less marked in suppurative than in rheumatic pericarditis, and it is often marked in rheumatism in which no pericarditis exists. We conclude, therefore, that the enfeeblement of the cardiac wall is mainly due to a direct toxic action of the rheumatic poison. However brought about, the dilatation is a most serious addition to the valvulitis or the pericarditis which may accompany it, and takes a very large share in the production of the dangerous symptoms usually attributed to them.

GROUP IV.—*The more chronic forms of chorea and rheumatism.*

Careful observation of the clinical course of these more chronic cases, and comparison with the results revealed by post-mortem examination, prove that in them also dilatation of the heart is one of the most important factors. A moderate valve lesion in a child is easily and effectually compensated if no fresh incidence of rheumatism occurs. On the other hand, grave symptoms of cardiac failure in a rheumatic child are almost always accompanied by fresh rheumatic manifestations. In 100 such cases ending fatally (Appendix C) there was evidence (apart from endocarditis) of fresh rheumatism in 86. We lay special stress on this fact, for it indicates that in children the fatal issue of rheumatic heart disease is not often the mechanical result of damaged valves, as is frequently the case in adults, and that some other explanation of the acute cardiac failure is required. And this is confirmed by the fact that the amount of change in the valves in such cases is usually moderate or slight. The chief cause of the fatal issue is indicated by the analysis of 150 autopsies in Appendix C, in which dilatation of the heart is specially mentioned in 92, while hypertrophy is noted in only 58.

The progress of rheumatism, as seen in hospital, fully supports this view. The cases which end fatally often present, during a course of several months, a series of rheumatic exacerbations,—now nodules, now arthritis, now pericarditis. The heart enlarges immensely, and the enlargement occurs synchronously with the rheumatic exacerbations. When they quiet down it ceases, and the area may even diminish. This enlargement is not a hypertrophy; the physical signs and the prostration of the patient prove that it is a heart-failure, and therefore a dilatation.

On the other hand, when no fresh rheumatic manifesta-

tion occurs, there is rarely any rapid increase of the cardiac area, even though valvulitis exists.

In chronic cases, then, if they are to prove fatal, the course is one of frequent rheumatic manifestations along with acute dilatations of the heart of greater or less severity.

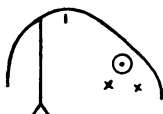
In illustration of the above remarks we now give tracings from two chronic cases.

CASE 9.—M. P—, aged 9, admitted into the Hospital for Sick Children, October 25th, 1895, under Dr. Penrose, for chorea. She remained in the hospital until November 30th, then was sent to the Highgate Convalescent Home, much better, but not well. She was readmitted February 6th, 1896, with pain in the side, and increase of the chorea, which had never disappeared.

M. P—, aged 9. Chronic chorea.



No. 1.  
Feb. 9, 1896.



No. 2.  
March 3, 1896.



No. 3.  
April 21, 1896.

! means accentuated pulmonary second sound. x x means impulse diffuse.

February 9th.—First tracing. Chorea severe. Area as shown.

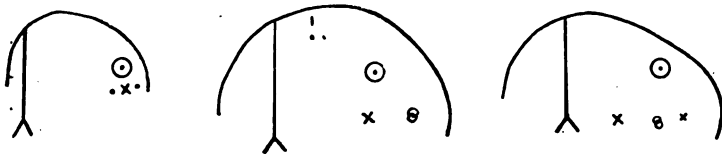
March 3rd.—Second tracing. Chorea still severe. A soft murmur now heard internal to nipple. Area further increased.

April 21st.—Third tracing. Chorea gone, after six months' duration. Area considerably diminished.

CASE 10.—W. M—, aged 8, admitted into the Hospital for Sick Children, December 4th, 1895, under Dr. Penrose, for chorea, endocarditis, and doubtful pericarditis; the attack dated from October.



W. M.—, aged 8. Subacute rheumatic fever, second attack.



No. 1.

Dec. 5, 1895.

No. 2.

March 26, 1896.

No. 3.

April 21, 1896.

! means accentuated pulmonary second sound. x means impulse.

8 means to-and-fro mitral murmur.

December 5th.—First tracing. Area as shown. Organic mitral murmur and doubtful pericardial friction.

Throughout December and January the chorea persisted, with slight irregular pyrexia; once in January what appeared to be pericardial friction was heard, and nodules were found. During the whole of February the child lay in bed, pale and speechless, with slight chorea; more nodules developed, and fresh pyrexia. Several cardiac tracings were taken.

March 26th, 1896.—Second tracing. Area much enlarged. Nodules still appearing.

During April he began slowly to improve, and the rheumatic symptoms to disappear.

April 21st.—Third tracing. Area diminishing.

The improvement continued, and he recovered sufficiently to leave the hospital.

Throughout the case all the symptoms pointed to dilatation of the heart, and not to hypertrophy or to pericardial effusion. It is extremely probable that the pericardium was completely adherent, for in 150 fatal cases, acute and chronic, it was found totally adherent in seventy-seven (see Appendix C). But it is doubtful how far even a completely adherent pericardium is in itself a cause of cardiac dilatation. In the records of St. Mary's Hospital we find thirty-four cases of entire adhesion of the pericardium discovered post mortem at ages between fifty and eighty-seven. (In none of these patients were

the kidneys granular.) Eighteen of these patients died from causes unconnected with the heart.

In bringing to a close our series of cardiac tracings, we would again draw attention to the evidence of the frequency and importance of dilatation afforded by the post-mortem records. We give brief notes of the condition found in three acutely fatal cases, from a list of fifteen such given in Appendix C.

1. F. G—, aged 7. Recent pericarditis, the pericardium partially adherent and granular. Marked dilatation. The mitral valve moderately thickened but functional. The aortic valves thickened. The myocardium pale.

2. T. B—, aged 10. Pericarditis with much recent adhesion and a little fluid. Fine granulations on the aortic valves and on the mitral flaps. All the cavities dilated. The muscle granular.

3. H. D—, aged 9. General pericarditis, recent and adhesive. Old vegetations on the mitral, but no stenosis. Aortic valves thickened. General dilatation.

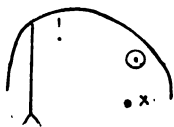
In each of these dilatation is a prominent feature, and valvular changes insignificant.

From the clinical records of these cases we find that death is often rapid, frequently sudden and unexpected, and preceded by pallor, collapse, vomiting, delirium, and restlessness, all pointing to grave circulatory failure. Active rheumatism is almost invariably present. Much excess of pericardial fluid is rare. Valvular change is usually moderate. Dilatation is more marked than hypertrophy.

The recognition of rheumatic dilatation may sometimes be of service in the diagnosis of an arthritis of otherwise doubtful nature. In July, 1896, a girl aged 11 was admitted into the Hospital for Sick Children under Mr. Morgan, for an acute arthritis of the right hip-joint. No other joint was affected. The thigh was swollen and excessively painful. Pyrexia was present. Acute tubercular disease of the joint was suspected, but the area of

cardiac dulness was enlarged, as seen in the subjoined tracing, and there was a soft systolic murmur external to

Girl aged 11. Rheumatic fever, first attack.



July, 1896.

! means accentuated pulmonary second sound. x means impulse.  
• means systolic murmur.

the apex. Extension was applied and salicylates given. Next day two interphalangeal joints of the toes of the left foot were swollen. The affection was undoubtedly rheumatic, and the child was rapidly and completely cured by salicylates. We are indebted to Mr. Morgan for permission to quote this case.

From the point of view of prognosis, the recognition of acute dilatation is of great importance. The occurrence of a second attack of rheumatism, attended by an acute dilatation, may entirely upset calculations founded merely on an estimation of the valvular damage remaining from the first attack. The possibility of a fresh rheumatic attack, causing increased dilatation, is by far the most important element in the prognosis.

Finally, with regard to treatment, we would point out that acute dilatation is often the earliest indication of a rheumatic heart affection. If, as we believe, salicylate of soda is a specific for rheumatism, and not a mere allayer of pain, it is clearly of the utmost importance to give this drug in sufficient doses as soon as the earliest indication of the pernicious action of rheumatism on the heart is manifested.

The recognition of dilatation will also make us lay greater stress on the necessity for rest in the treatment both of rheumatism and of chorea. And when the question of the advisability of paracentesis pericardii is being

considered, the remembrance of the frequency of marked dilatation and of the rarity of much excess of fluid in the pericardial cavity may save us from a therapeutic disaster which has befallen more than one skilled physician in the past.

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#### APPENDIX A.

*Observations upon the condition of the heart in forty-five children suffering from surgical ailments of slight severity, and free from any disease of the chest. These observations were made at the Hospital for Sick Children, Great Ormond Street, May to November, 1896.*

The exact method adopted was as follows:—A tracing of the deep cardiac dulness was made in every case. The child was previously kept in bed for a day at least after admission to the hospital. The dulness was percussed from the left side, the patient lying on the back with the arm abducted and chest quite uncovered. The area was marked out as delineated with a dermatographical pencil; the impulse, left nipple, and mid-sternum were indicated, together with the costal angle. The condition of the sounds and any peculiarity of the chest or physique were also recorded. Finally, the outline was at once transferred to tracing paper and dated.

The results obtained are as follows:

#### I. The ages of the children:

Between 11 and 12 years	.	.	10 cases.
„ 10 „ 11 „	.	.	6 „
„ 9 „ 10 „	.	.	5 „
„ 8 „ 9 „	.	.	5 „
„ 7 „ 8 „	.	.	3 „
„ 6 „ 7 „	.	.	8 „
„ 5 „ 6 „	.	.	2 „
„ 4 „ 5 „	.	.	1 case.
„ 3 „ 4 „	.	.	3 cases.

Between 2 and 3 years . . .	1 case.
„ 1 „ 2 „ . . .	1 „

---

45

## II. The particular surgical ailments:

Early morbus coxæ . . . . .	5
Hydrocele . . . . .	3
Rectal polypus . . . . .	1
Early tubercular knee . . . . .	2
Congenital torticollis . . . . .	2
Early spinal caries . . . . .	1
Radical operations for hernia . . . . .	1
Cleft palate . . . . .	4
Rickets . . . . .	3
Functional affection . . . . .	1
Tubercular glands . . . . .	4
Adhesions in elbow-joint . . . . .	4
Chronic abscess . . . . .	4
Old excision of knee . . . . .	1
Lupus of skin . . . . .	1
Gummata over clavicle . . . . .	2
Ranula . . . . .	1
Talipes . . . . .	2
Nævus . . . . .	1
Hammer-toe . . . . .	1
Fracture . . . . .	1

---

Total . . . 45

## III. Distances from the mid-sternum to the nipple.

Ages: 11—12, from  $2\frac{3}{4}$  to  $3\frac{1}{2}$  inches.

10—11, „ $2\frac{1}{2}$ „ 3 „
9—10, „ $2\frac{1}{2}$ „ 3 „
8—9, „ $2\frac{1}{2}$ „ 3 „
7—8, „ $2\frac{1}{2}$ „ $2\frac{3}{4}$ „
6—7, „ $2\frac{1}{4}$ „ $2\frac{3}{4}$ „
5—6, „ $2\frac{1}{4}$ „ $2\frac{1}{2}$ „
1—5, „ $2\frac{1}{4}$ „ $2\frac{1}{2}$ „

# ACUTE DILATATION OF THE HEART

ne drawn at a right angle to the mid-sternal line  
the most limit of the tracing passed through the—

First rib on the left side in . . .	37 cases.
Second space „ „ . . .	7 „
Third space „ „ . . .	1 case.

---

45

V. The distance from the mid-sternum to the furthest  
of the heart to the right:

$\frac{1}{4}$ inch in . . . . .	2 cases.
$\frac{1}{2}$ „ . . . . .	19 „
$\frac{3}{4}$ „ . . . . .	13 „
1 „ . . . . .	8 „
$1\frac{1}{4}$ inches in . . . . .	1 case.
Doubtful in . . . . .	2 cases.

---

45

The right limit of the heart is not always easily ascer-  
tained, either because the chest is misshapen or because  
there is considerable emphysema. These difficulties are,  
however, exceptional; if the chest is misshapen it may  
be difficult to ascertain any limit of the cardiac dulness.

The right limit of the cardiac dulness in children is  
normally about one fingerbreadth to the right of the right  
margin of the sternum, as ascertained by percussion.  
But as this limit is traced from above downward it tends  
inward a little toward the middle line, following the  
curve of the auricle. In the healthy heart this is a  
difficult point to ascertain. When the heart is dilated it  
is more easily appreciated, but even then may be difficult  
to demonstrate with certainty.

VI. The relation of the left limit of the cardiac dulness  
to the left vertical nipple-line:

Internal to the nipple in . . .	19 cases.
In the nipple line in . . .	18 „
External to the nipple in . . .	8 „

---

45

Six of the 8 cases in which the dulness extended outside the nipple line were under six years of age. The exact measurements in the eight cases were—

$\frac{1}{4}$ inch in	.	.	.	.	4
$\frac{3}{8}$ „	.	.	.	.	2
$\frac{1}{2}$ „	.	.	.	.	2
					—
					8

In 3 of these cases the children appeared normal, in 3 the chest was noted as small and narrow, in 2 the children were weak and delicate.

It should be stated that in several cases a small, narrow chest was noted, but the left limit was internal to the nipple-line.

VII. The relation of the impulse to the cardiac dulness. This is often considerably internal to the left limit of the cardiac dulness.

Thus, of 25 cases, in 17 it was internal to this left limit, in 2 it was traced outward as far as the limit, in 6 it was absent to touch.

VIII. The sounds.

The first sound was usually distinctly longer than the second sound. The second sound to the left of the sternum at the base was usually louder than the second sound in the second right space.

In twelve cases the greater loudness of the pulmonary second sound was especially noted.

IX. Irregularity of the heart was exceptional.

X. Bruits were found in some cases.

In 10 an occasional soft systolic murmur was heard in the fourth space just to the left of the sternum.

In 6 a systolic murmur was heard at the base on the left side.

## APPENDIX B.

*Observations upon the hearts of 35 healthy public-school-boys between the ages of twelve and fourteen.*

We realised that cases in the surgical wards of an hospital, though free from chest affections, are not perfectly healthy children. We have accordingly supplemented the results given in Appendix A with the following observations upon healthy schoolboys at Marlborough College. We are much indebted for permission to Dr. Penny, Medical Officer to the school.

In these cases tracings were not taken, but the distances were noted as each cardiac area was percussed. The order of examination, as in previous cases, was firstly, palpation; secondly, percussion; thirdly, auscultation. The same posture, namely, the supine with the left arm abducted, was adopted as in the previous investigations in Appendix A.

The results obtained were as follows:

I. The ages: between twelve and thirteen years, 4; between thirteen and fourteen years, 31.

II. The action of the heart was regular, except in the case of one nervous boy. In this case the irregularity soon passed off.

III. The position of the impulse in relation to the vertical nipple-line and to the ribs:

In the fifth space in . . .	23 cases
At the fifth rib in . . .	6 „
In the fourth space in . . .	4 „
Not localised in . . .	2 „

The impulse was internal to the vertical nipple-line in 32 cases, and frequently considerably so.

Of the 3 cases in which the impulse was felt in the vertical nipple-line, in 1 case the boy was delicate and in the doctor's house, though in good health at the time.

Case 2 and Case 3 had suffered from influenza six weeks before. This fact was of especial interest, because



the condition of the heart had been ascertained in entire ignorance of this history of influenza.

#### IV. The deep cardiac dulness.

(A) The left limit: in these boys with well-formed chests this area was obtained with considerable ease.

(a) In 21 cases the left limit was about one inch internal to the nipple.

(b) In 7 cases it reached a vertical line through the inner margin of the areola.

(c) In 5 cases it reached the vertical nipple-line.

(d) In 2 cases it reached external to the vertical nipple-line.

i. Of these two latter cases, in one the limit extended  $\frac{3}{4}$  inch external, in one the limit extended 1 inch external.

One of these two boys was the delicate boy already mentioned, and in his case there were also soft systolic murmurs at the base and apex.

The other boy had suffered from an attack of influenza six weeks previously; he had also a soft systolic murmur in the fourth space internal to the nipple.

ii. Of the 5 cases in which the dulness reached the vertical nipple-line, in each there was a soft systolic murmur internal to the nipple.

iii. Of the 7 cases in which the dulness extended almost to the vertical nipple-line, in four there was a soft systolic murmur internal to the nipple, and in one the first sound was slightly blurred.

iv. Of the 21 cases in which the dulness at its left limit was distinctly internal to the vertical nipple-line, in only two a soft systolic murmur was noted internal to the nipple.

(B) The right limit: in 4 cases hyper-resonance prevented the limit being defined.

In 2 cases the limit was  $1\frac{1}{4}$  inches from the mid-sternum.

In 29 cases the limit was from  $\frac{3}{4}$  to 1 inch to the right of the mid-sternum.

(c) The upward limit to the left of the sternum.

The third rib in 34 cases, the second space in 1 case.

V. The sounds.

In 5 cases the first sound at the apex was rather short.

The second sound over the left second space was almost invariably louder than over the right second space, but both were well defined and sharp.

In the cases with soft systolic murmurs the second sound to the left was especially louder than that to the right.

VI. Murmurs. There was not a musical murmur in any of these cases, they were all soft blowing murmurs, some diminished by the erect posture.

The situation in which they were heard most frequently and at their loudest was in the fourth space close to the left margin of the sternum. This murmur became fainter when traced to the nipple, and in only one case was heard external to it, and then very faintly indeed. It also became fainter when traced toward the base.

The exact numbers in which murmurs were heard when classified are as follows :

Blowing murmurs were heard in 15 cases.

In 9 cases out of this number the murmur was best heard in the fourth space on the left side.

In 6 cases the murmur was basal.

In three of the 15 cases a murmur was heard in both situations.

In each of these three cases the cardiac dulness was more extensive than usual.

#### APPENDIX C.

*An analysis of 150 fatal cases of rheumatic morbus cordis in children, derived from the post-mortem records of the Hospital for Sick Children, Great Ormond Street, and St. Mary's Hospital.*

The analysis contains observations upon the following points :

1. The sex incidence (150 cases).
2. The age incidence (150 cases).
3. The number of fatal first attacks (115 cases).
4. The relation of the attacks to scarlet fever (100 cases).
5. The season in which the fatal attack commenced (150 cases).
6. The condition of the pericardium (150 cases).
  - (a) As to adhesion.
  - (b) As to fluid.
7. The condition of the myocardium (150 cases).
8. The condition of the mitral valve (150 cases).
9. The condition of the aortic valve (150 cases).
10. The condition of the tricuspid valve (150 cases).
11. The condition of the pulmonary valve (100 cases).
12. The combination of valvular lesions (150 cases).
13. The evidence of fresh rheumatic manifestations in the fatal illness (150 cases).
14. The frequency of nodules in fatal cases (87 cases).
15. The frequency of pericardial friction toward the end of the illness (100 cases).
16. The frequency of marked dropsy (100 cases).
17. The condition of the heart in 25 cases with marked dropsy.
18. The frequency of sudden death as the termination of the illness (100 cases).
19. The frequency of chorea in the last illness (100 cases).
20. The condition of the heart *post mortem* in fifteen acute attacks of rheumatic fever rapidly fatal.

#### I. The sex :

Females . . . 88 = 59 per cent.

Males . . . . 62 = 41 „

#### II. The age incidence :

Up to  $3\frac{1}{2}$  years of age 1 case = 0.6 per cent.

$3\frac{1}{2}$  „  $4\frac{1}{2}$  „ „ 6 cases = 3.9 „

$4\frac{1}{2}$  „  $5\frac{1}{2}$  „ „ 11 „ = 7.4 „

5½ to 6½ years of age	14 cases	=	9·4 per cent.
6½ „ 7½ „ „	19 „	=	12·7 „
7½ „ 8½ „ „	19 „	=	12·7 „
8½ „ 9½ „ „	23 „	=	15·3 „
9½ „ 10½ „ „	30 „	=	20 „
10½ „ 11½ „ „	18 „	=	12 „
11½ „ 12 „ „	9 „	=	6 „

---

150

The maximum is reached at the tenth year.

### III. Fatal first attacks.

It is not always easy to be certain that the attack which proved fatal was really the first; but out of 115 fatal cases 35 were apparently first attacks.

### IV. The relation (if any) to scarlet fever.

Of 100 cases of fatal rheumatism, in which the occurrence or non-occurrence of scarlet fever was noted, in only six did there seem to be any possibility of a relation between the two diseases. The relation in these six cases was as follows:

In one, scarlet fever occurred three years before, "child never well since."

In one, scarlet fever occurred ten weeks before the symptoms of rheumatic fever.

In one, scarlet fever occurring two years before was at once followed by an attack of rheumatic fever.

In one, scarlet fever was followed in a week by rheumatic fever.

In one, scarlet fever was followed in a month by rheumatic fever.

In one, scarlet fever was "followed shortly" by rheumatic fever.

It appears, therefore, that rheumatic fever in the great majority of cases arises quite apart from scarlet fever.

### V. The season in which the fatal attack commenced.

This analysis is founded on the appearance of the first

symptoms of the illness. This date must be open to some doubt, but in a large number of observations should be fairly accurate.

Of 150 cases 67 commenced in the autumn.

33	„	spring.
30	„	winter.
20	„	summer.

That is, 66 per cent. commenced in autumn and spring.

34 „ „ winter and summer.

#### VI. The condition of the pericardium in 150 cases :

##### (A) As to adhesions :

- i. More or less adherent in 113 = 75 per cent.
- ii. Not adherent in . . . 37 = 25 „
- i. In the cases (113 in number) of adhesion—
  - (a) In 77 the adhesion was general.
  - (b) In 36 it was partial.
    - (a) In the 77 cases of total adhesion—
      - 19 were stated to be old adhesions.
      - 18 „ „ recent adhesions.
      - 40 were of doubtful age.
    - (b) In the 36 cases of partial adhesion—
      - 6 were described as adherent.
      - 8 „ „ as much adherent.
      - 13 „ „ as partly adherent.
      - 9 showed recent granular pericarditis.
  - ii. In the 37 cases described as not adherent, in only 9 was the pericardium definitely stated to be healthy.

##### (B) As to fluid in the pericardial cavity :

In 38 cases out of 150 = 25 per cent., a special note was made of fluid in the pericardial sac in the following terms :

“Little” or “very little”	.	in 9 cases.
“Some”	.	in 3 „
“Under one ounce”	.	in 4 „
“Between one and two ounces”	in 6	„
“Between two and three ounces”	in 6	„
“Five ounces”	.	in 1 case.

"Six ounces"	..	in 1 case.
"Excess"	.	in 4 cases.
"Much excess"	.	in 4 ,,

---

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Thus in only 12 out of 150 cases of fatal rheumatic fever is it definitely stated that more than *two* ounces of fluid were found in the pericardial cavity, and in only 6 cases more than *three* ounces.

It is worthy of careful note that much fluid in the pericardium is rarely found after death from rheumatic pericarditis.

#### VII. The condition of the myocardium in 150 cases :

(A) As to change in appearance, macroscopic and microscopic.

In 34 cases there is special mention of change—that is in 23 per cent.—in the following terms :

"Soft and pale"	.	in 15 cases.
"Tough and fibroid"	.	in 8 ,,
"Fatty"	.	in 4 ,,
"Opaque"	.	in 1 case.
"Striæ indistinct"	.	in 1 ,,
"Myocardial change"	.	in 5 cases.

These numbers can only be looked upon as evidence that myocardial change occurs. They are no gauge of the frequency of its occurrence. In many of the cases microscopic examinations of the cardiac walls were not made.

(B) The condition of the myocardium as to hypertrophy and dilatation in 150 cases.

*Hypertrophy* is especially mentioned in 58 cases in the following terms :

- i. "Great," "much," or "general" hypertrophy in 18 cases. In two of these the left ventricle was especially affected.
- ii. "Hypertrophy" in 29 cases.  
Of these, in 7 the left ventricle especially.  
in 1 the right ventricle especially.

iii. "Some hypertrophy" in 8 cases.

iv. "A little hypertrophy" in 3 cases.

*Dilatation* is especially mentioned in 92 cases. Thus:

i. "Marked," "much," or "general" dilatation in 56.

Of these—

(a) "General dilatation" . in 19 cases.

(b) "Marked" and "much" in 24 ,,

(c) "Enormous" . in 1 case.

(d) "Great" . in 5 cases.

(e) "Of both ventricles" . in 5 ,,

(f) "Of the left ventricle" . in 1 case.

(g) "Of the right ventricle" in 1 ,,

ii. "Dilatation especially" in 4 cases.

iii. "Dilatation" in 24 cases.

iv. "Some dilatation" in 6 cases.

v. "Walls thin" in 2 cases.

In forming from these data an opinion as to the frequency and relative amount of hypertrophy and dilatation respectively, it must be remembered that in some of the cases no definite statement is made; that in others a preponderance of hypertrophy may have caused the omission of any statement as to dilatation, or *vice versa*; finally, that the very existence of slight degrees of hypertrophy or dilatation may be differently judged by different observers. For these reasons the numbers have not been reduced to percentages, but the general tendency of the facts recorded seems to indicate that dilatation is both more frequent and more marked than hypertrophy. For while the latter is noted in 58 cases the former is noted in 92, and in 56 of these it was evidently a striking fact.

VIII. The condition of the mitral valve in 150 cases. Affected in 149.

The following are the descriptions of the changes:

(1) "Marked mitral stenosis" in . 9 cases.

(2) "Marked mitral regurgitation"

in . . . 11 ,,

(3) "Much thickening and puckering" in . . .	8 cases.
(4) "Mitral regurgitation" } "Mitral stenosis" } "Double mitral" }	in . 41 ,,
(5) "Some thickening" in . . .	45 ,,
(6) "Numerous vegetations" in . . .	4 ,,
(7) "Vegetations and small deposits" in . . .	31 ,,

---

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We may conclude that some affection of the mitral valve is almost invariably present in fatal rheumatism in children; that in about one half of the fatal cases the lesion is slight, in about one fifth great; that thickening of the mitral valve is common, but that a marked degree of stenosis is rare in children under twelve.

IX. The condition of the aortic valves in 150 cases. The valve was affected in 51 cases = 34 per cent.

The details are as follows:

(1) "Much aortic regurgitation" in . . .	1 case.
(2) "Definite aortic regurgitation" in . . .	4 cases.
(3) "Definite aortic stenosis" in . . .	4 ,,
(4) "Vegetations and beading" in . . .	19 ,,
(5) "Thickening" in . . .	18 ,,
(6) "Aortic valves affected" in . . .	5 ,,

---

51

Slight change is the rule.

X. The condition of the tricuspid valve in 150 cases. This valve was affected in 36 cases = 24 per cent.

The details are as follows:

"Marked stenosis" in . . .	1 case.
"Vegetations" in . . .	15 cases.
"Thickening" in . . .	20 ,,



Again, if the valve is affected at all, slight change is the rule.

XI. The condition of the pulmonary valve in 100 cases.

This was affected in 4 = 4 per cent., as follows :

“ Thickened ” . . . . in 3 cases.

“ Dilated ” . . . . in 1 „

XII. The frequency of a combination of valvular lesions in 149 cases.

(a) In 4 cases at least all the four valves were affected.

(b) In 32 cases the mitral, aortic, and tricuspid valves were affected.

(c) In 15 the mitral and aortic valves were affected.

(d) In 98 the mitral valve only was affected.

XIII. The evidence of fresh rheumatic manifestations in 100 fatal cases.

The evidence relied upon consists of chorea, pericarditis, nodules, arthritis, and erythema occurring during the fatal illness.

The possibility of fresh endocarditis supervening upon old is not taken into account because of the difficulties in proving its occurrence.

Eighty-six out of 100 cases showed undoubted manifestations of recent rheumatism.

Of the remainder = 14 per cent.—

Three cases were doubtful.

Two cases died with much cardiac dilatation.

One case died with cerebral hæmorrhage.

One case died, and total adhesion of the pericardium and much endocarditis were found.

Two cases died with “ marked mitral stenosis.”

Two cases died with “ marked mitral regurgitation.”

Two cases died, and a totally adherent pericardium was found.

In one case there was a history of three years' gradual failure.

These figures are worthy of especial note, for they

point strongly to this fact, that cardiac break-down in children with damaged hearts is mainly due to a fresh rheumatic attack, not to overstrain.

XIV. The frequency of nodules in fatal cases since 1879.

Eighty-seven cases are recorded since that date. In 47 of these nodules were present, and in 8 their occurrence was doubtful. That is, nodules are found in about 54 per cent. of fatal cases.

This result probably rather under-estimates the frequency of their occurrence.

XV. The frequency of pericardial friction toward the end of the fatal illness.

Of 100 cases, in 55 friction was noted, in 15 it was doubtful, and in 30 it was not mentioned.

XVI. The frequency of marked dropsy.

Observations are taken from 100 cases.

In 25 cases it was described as "much" or "considerable."

In these cases—

- (a) It was of long standing in 8 cases.
- (b) It was of some standing in 8 cases.
- (c) Quite recent in 9 cases.

XVII. The condition of the heart as found post mortem in these 25 cases of marked dropsy.

Group A.—The 8 cases of long standing.

- (1) In 3 there was marked mitral stenosis.
- (2) In 1 there was thickening of the mitral valve, and the pericardium was  $\frac{1}{4}$  of an inch thick.
- (3) In 1 there was mitral stenosis and incompetence, and aortic regurgitation.
- (4) In 1 there was marked mitral and tricuspid incompetence.
- (5) In 2 mitral incompetence.

Group B.—Eight cases of dropsy of some standing.

- (1) In 1 there was marked mitral stenosis.
- (2) In 3 there was marked mitral incompetence.

- (3) In 2 the heart was described as fibroid, and the pericardium was totally adherent.
- (4) In 1 the chordæ tendineæ of the mitral valve were described as much thickened.
- (5) In 1 the pericardium was adherent to the chest wall.

The chief feature in these cases of marked dropsy is the occurrence of decided valvular disease.

XVIII. The frequency of sudden death in 100 cases ; by this is meant the occurrence of death unexpectedly in the last illness. This occurred in 33 cases, as follows :

- (a) "Very sudden" in 11. In three of these myocardial change was noted.

In two of these cases it is expressly stated that just before the sudden end the children had been improving.

- (b) Attacks of collapse or much vomiting, then a sudden end, in 8 cases.

- (c) "Sudden" in 8 cases.

- (d) "Rather sudden" in 6 cases.

In many more cases there was severe vomiting some days before death.

XIX. Fatal cases in which chorea was a prominent symptom. Some of these cases had other evidences of severe rheumatism at the time of admission, others rapidly developed them. Twenty cases are recorded here, and 14 of these were first attacks of rheumatism.

This is very striking confirmation of the close association of rheumatism and chorea—rheumatism, too, which may prove to be of the most virulent type.

XX. Brief abstracts of the post-mortem appearances of the heart in fifteen acute cases rapidly fatal.

CASE 1.—E. K—, aged 11. Chorea and recent rheumatic fever.

*Post-mortem.*—The pericardium contained a considerable amount of fluid, with flakes of lymph. The heart weighed 8½ ounces. There were rows of bead-like granulations on the mitral, aortic, and tricuspid valves.

CASE 2.—M. W—, aged 9. Chorea, first attack, and rheumatic fever.

*Post-mortem.*—Recent granular pericarditis, a little fluid, much dilatation of all the cavities, and recent vegetations on the mitral and aortic valves. The heart muscle was flabby.

CASE 3.—G. K—, aged 11. Acute rheumatic fever.

*Post-mortem.*—Recent pericarditis with extensive lymph-shreds, dilatation, but very little hypertrophy of the ventricle. The mitral valve thickened and fringed with vegetations. Minute vegetations on the aortic and tricuspid flaps.

CASE 4.—F. S—, aged 3. Acute rheumatic fever.

*Post-mortem.*—Recent pericarditis; a few drachms of fluid in the sac. Much general dilatation. The mitral valve dilated, with recent vegetations on the mitral and tricuspid flaps.

CASE 5.—J. B—, aged 10. Acute rheumatic fever.

*Post-mortem.*—Pericarditis with much recent adhesion and a little fluid; fine aortic granulations, and some fine granules on the mitral flaps. All the cavities dilated. muscle granular.

CASE 6.—F. J—, aged 4. Acute rheumatic fever.

*Post-mortem.*—Recent pericarditis; a little fluid. Much dilatation of all the cavities. Some mitral thickening and a few vegetations on the aortic valves.

CASE 7.—P. W—, aged 3½. Acute rheumatic fever.

*Post-mortem.*—Recent pericarditis with adhesion; one ounce of fluid in the sac. Heart dilated. Slight vegetations on the tricuspid flaps. Numerous fine vegetations on the mitral.

CASE 8.—M. M—, aged 6½. Acute rheumatic fever.

*Post-mortem.*—Recent general adhesive pericarditis. Two drachms of fluid in the sac. General dilatation with a little hypertrophy of the left ventricle. Some old thickening of the mitral valve. Slight aortic puckering.

CASE 9.—H. W—, aged 9. Acute rheumatic fever.

*Post-mortem.*—Pericarditis, general, recent, and adhesive. Old vegetations on the mitral, but no stenosis. The aortic valves thickened; general dilatation of cavities.

CASE 10.—M. W—, aged 8. Acute rheumatic fever.

*Post-mortem.*—General pericarditis. Some fluid in the pericardial sac. Dilatation of the ventricles. Some hypertrophy. Beading of the mitral valve.

CASE 11.—C. S—, aged 8. Rheumatic fever and chorea.

*Post-mortem.*—Recent pericarditis, general and adhesive. Small vegetations on the aortic and mitral valves; heart enlarged.

CASE 12.—F. B—, aged 8. Acute rheumatic fever.

*Post-mortem.*—Pericardium totally adherent; recent adhesions. General dilatation, little valvular change.

CASE 13.—G. N—, aged 6. Acute rheumatic fever.

*Post-mortem.*—The pericardium contained two ounces and a half of fluid with recent flakes. Much dilatation, and dilatation of the valve orifices. Little valvular change. Fatty changes in the heart muscle.

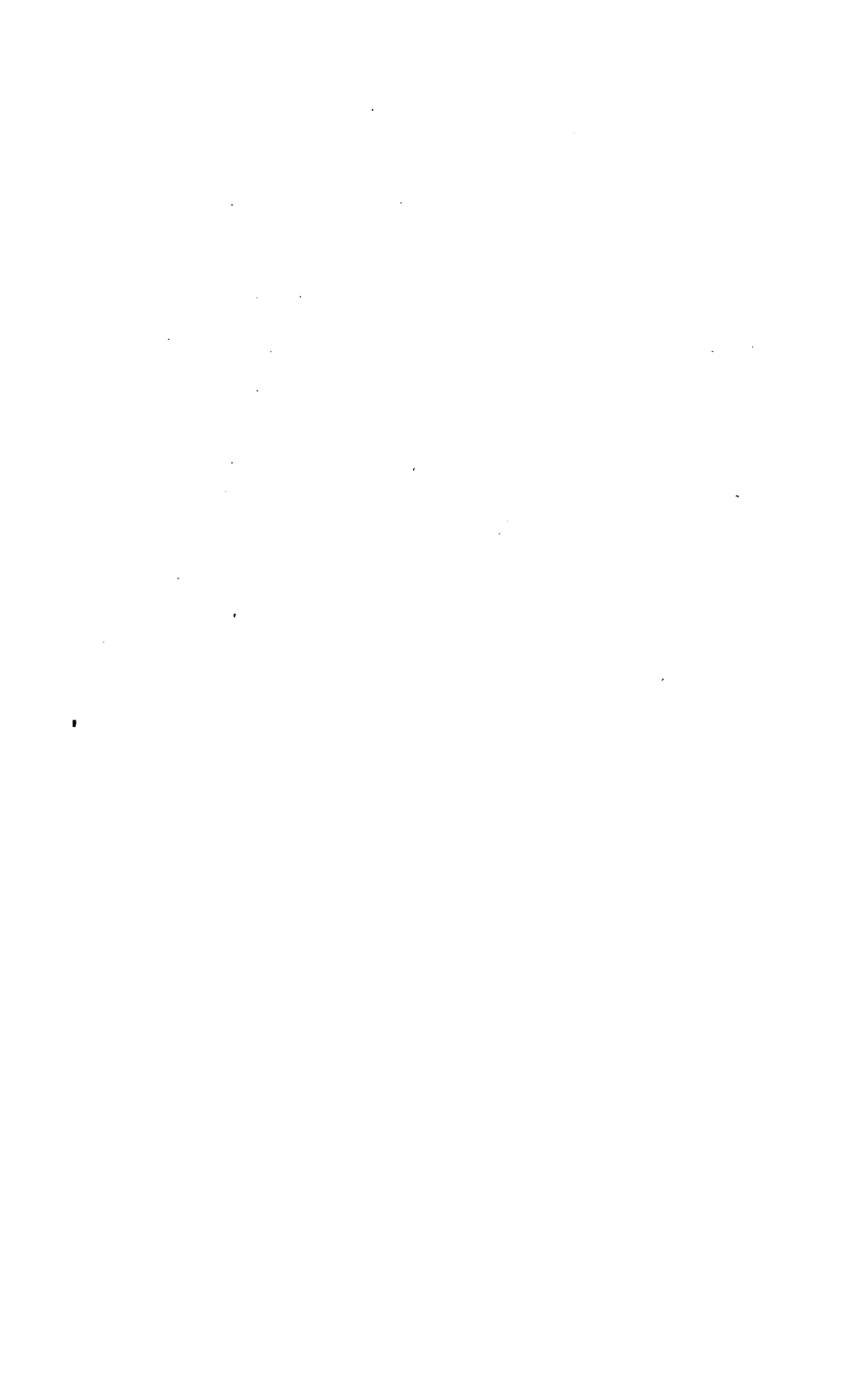
CASE 14.—H. B—, aged 11. Acute rheumatic fever.

*Post-mortem.*—Pericarditis, with recent adhesions to the left ventricle of the heart. Some mitral thickening, much general dilatation, and dilatation of the valve rings. Fatty changes in the heart muscle.

CASE 15.—A. B—, aged 11. Acute rheumatic fever.

*Post-mortem.*—The pericardium contained one ounce and a half of fluid. Some adhesions, no lymph. Mitral and tricuspid beading.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 189.)



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*The Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted in the Library copy, where the entire Index to the current date may always be consulted.*

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